ARCHIVES OF OTOLARYNGOLOGY

VOLUME 38 NOVEMBER 1943 NUMBER 5

COPYRIGHT, 1943, BY THE AMERICAN MEDICAL ASSOCIATION

TRAUMA OF THE LARYNX

CHEVALIER JACKSON, M.D.
AND
CHEVALIER L. JACKSON, M.D.
PHILADELPHIA

In this paper we present the results of clinical observations on industrial, criminal, accidental and suicidal trauma of the larynx. Their publication seems especially called for because of the dearth of literature on the subject. With a single exception [1] no textbook on the throat gives any consideration to the subject of laryngeal trauma. Textbooks dealing with traumatic surgery in general mention the larynx briefly if at all. This neglect of the subject is curious when we consider that the larynx occupies a position of utmost importance on both the absolutely vital "lines of communication: the air and food passages." [1] The accepted idea that laryngeal injury is rare may have something to do with the paucity of literature. Undoubtedly the rarity of incidence has been exaggerated, but relative infrequency makes it all the more necessary that the fundamentals of treatment be generally well known, and this is particularly necessary in cases of laryngeal involvement because of the importance of the nine functions of the larynx [1] as well as because of its vital location in the air and food passages.

So important is trauma of the larynx in the treatment of injuries of the neck that such injuries might well be divided into two classes according to whether the larynx is involved or not.

INCIDENCE

Surgical statistics give no indication whatever of the frequence of anatomic trauma of the larynx; in fact, they are directly misleading, because practically all of the cases of laryngeal trauma have been recorded as cases of injury of the neck. Structural injury of the larynx without any injury of the neck is not common; injury of the larynx along with other cervical tissues is certainly not infrequent, and damage to the exterior of the larynx is not rare in trauma of the neck.

Mortality statistics afford no data whatever on deaths from asphyxia or hemic drownage in cases of cervical trauma involving the larynx under conditions in which immediate surgical attention is impossible.

ETIOLOGY

The larynx is protected posteriorly by the cervical part of the spinal column and above by the skull; in front it is partially protected by the projection of the chin and the anterior wall of the thorax. Still more is it protected by its resilient suspension. It is widely exposed on both sides, and in front it is exposed to being injured by any object of such shape or position that it can get between the chin

From the Department of Broncho-Esophagology, Temple University School of Medicine.

1. Jackson, C., and Jackson, C. L.: Diseases and Injuries of the Larynx, ed. 2, New York, The Macmillan Company, 1942.

and the thorax. As a matter of fact the protection of the larynx and its escape from injury are both exaggerated in an effort to explain rarity in the records which, as stated, is really due to the fact that nearly all cases of laryngeal injury are recorded under the general classification of injuries of the neck.

Available records of personal experience show that the causes of laryngeal trauma have been classifiable as follows:[2]

1. Highway accidents: (*a*) gash by shattered windshield; (*b*) contusion by striking of neck on rim of steering wheel in collision; (*c*) fracture of thyroid cartilage by striking of neck on unknown part of wreckage.

2. Railway accidents: Train crew: (*a*) catching of neck on a sagged wire; (*b*) crushing of neck in hand-coupling of cars; (*c*) striking of neck by handle bar of handcar. Passenger: (*d*) striking of neck on back of forward car seat in collision.

3. Industrial accidents: (*a*) burn by live electric wire; (*b*) premature blast in quarry; (*c*) fall on moving gears while intoxicated; (*d*) striking of neck on end of pick handle when stooping in dark; (*e*) burns from inhalation of chemical fumes and hot smoke.

4. Household accidents: (*a*) fall with neck striking edge of step, (*b*) table, (*c*) chair, (*d*) step-ladder; (*e*) burns from swallowed caustic agent (lye).

5. Farm accidents: (*a*) kick of a horse; (*b*) trampling by horse's hoof.

6. Medical and surgical accidents: (*a*) stabbing tracheotomy; (*b*) misdirected intubation tube; (*c*) aggressive endolaryngeal surgery[3]; (*d*) radium and roentgen ray overdosage; (*e*) forceps delivery.

7. Sporting accidents: (*a*) trampling by football player; (*b*) stroke of golf ball; (*c*) baseball; (*d*) striking neck on sled while coasting.

8. Brawls: (*a*) blow of fist; (*b*) gash with backed razor; (*c*) stab with ice pick; (*d*) gash with cleaver; (*e*) gash with butcher knife.

9. Police encounters: (*a*) perforation by bullet; (*b*) gash with knife; (*c*) blow with lead pipe.

10. Suicidal attempts: (*a*) crushing and displacing compression by hanging; (*b*) gash with razor; (*c*) forcing of two foreign bodies, a key and a screweye into the larynx.

PATHOLOGIC CONSIDERATIONS

Occlusion of the lumen of the larynx may occur immediately without inflammation. The fundamental anatomic fact is that the lumen of the larynx is normally not much larger than is required for respiration. Therefore relatively little encroachment on the lumen is a serious matter and no great mass is required to produce asphyxia. Sufficient tissue to occlude the lumen may be displaced at the moment of injury, resulting in immediate asphyxia. Occlusion may come on in a few minutes from submucosal hemorrhage, a hematoma. An hour or more later the lumen may be occluded by submucosal emphysema. This may be accompanied by mediastinal emphysema; aided by muscular pressure and thoracic suction, the air quickly finds its way downward between the layers of cervical tissue. Subcutaneous emphysema may or may not coexist. Simultaneous wounding of adjacent large vessels may produce a flow of blood large enough to enter the trachea and drown the patient.

The foregoing processes are *noninflammatory* and must be borne in mind as immediate emergencies. Inflammatory reaction developing later may cause occlusion of the laryngeal lumen by the well known "edema of the glottic tissues."

As with traumatic lesions elsewhere, a wound of the larynx may be in the form of a crush, a contusion, an incision, a laceration, a puncture, a penetration,

2. (*a*) Jackson, C. L.: Trauma of the Larynx, S. Clin. North America **19**:1479-1487 (Dec.) 1939. (*b*) Jackson, C.: Stenosis of the Larynx with Special Reference to Curative Treatment with Core Molds, Tr. Am. Laryng., Rhin. & Otol. Soc. **42**:12, 1936. (*c*) Jackson and Jackson.[1]

3. Imperatori, C. J.: Presentation of a Patient, Tr. Am. Laryng. A. **61**:257, 1939; Subsequent Report of a Patient with Laryngeal Stenosis First Reported in 1939, ibid. **62**:47, 1940.

a perforation, a fracture or a dislocation. The traumatizing agent may remain, may have passed on or may have been withdrawn; in either of the latter two happenings it may or may not have left extraneous matter.

There may be associated injury of adjacent tissues, or the larynx may be severely injured with no evidence of any other trauma nearer than the shoulder. Traumatic laryngectomy with survival has been reported. In a case in our experience the entire larynx of a quarryman was torn away by a premature blast. Doubtless there are many fatal cases in which the larynx has been destroyed along with a part of the neck.

Detailed consideration of these various wounds has been given elsewhere.[1]

It seems proper here to emphasize again the importance of recognizing three classes of cases peculiar to the larynx, namely, (1) those in which the cartilage and its perichondrium are uninjured, (2) those in which these structures are damaged and (3) those in which there is involvement of the hypopharynx or of the cervical part of the esophagus. Other phases being equal, prognosis and treatment in any given case are dependent on the determination of which one of these three classes covers the particular case. The pathologic course, clinical features and duration in each class are quite different.

If the cartilage and its perichondrium have not been injured, recovery is usually prompt, and complications, if any, are only those of any wound.

If, on the other hand, the cartilage and its perichondrium are involved, there is almost certain to be perichondritis, prolonged suppuration, chondral necrosis and building of cicatricial tissue whose subsequent contraction unresisted by a cartilaginous framework ends in chronic laryngeal stenosis, even total atresia. Reparative processes of cartilage anywhere are poor, poorer even than those of bone. In laryngeal cartilages of adults union is fibrous and rarely, if ever, cartilaginous.

In the third class of cases, those in which there is an opening from the wound into the hypopharynx or the cervical part of the esophagus, the wound is deluged with oral secretions, food and fluids, containing all the infective agents of the mouth, tonsils, nasal passages and accessory cavities. If there have been extensive lacerations, the wound may be covered with grayish sloughs similar to those usually formed after tonsillectomy. Unless controlled by antiseptics, such a wound is a foul mess.

Fracture, if due to garroting or to a blow, usually involves the partially ossified anterior part of the thyroid cartilage where the wings meet; the superior horns may be broken off. This form of injury is often accompanied by fracture of the hyoid bone; the cricoid cartilage, which is a complete firm ring, is more resistant. Missiles, penetrating or perforating, may shatter any of these structures, or may only plough a groove in the external surface. In a case in our experience, that of a policeman, both arytenoid cartilages were shot away by a perforating bullet. Any injury to laryngeal cartilage may or may not be followed by emphysema but is almost invariably followed by perichondritis, chondral necrosis, prolonged suppuration and, if not prevented, cicatricial stenosis or even total atresia.

Injury to the inferior laryngeal nerve in trauma of the neck, causing abductor paralysis without direct injury to the larynx itself, is rare, but we have seen 2 cases. In one the injury was unilateral; in the other, bilateral.

Dislocation occurs from an extremely forcible displacement of the cricoid cartilage backward alongside the vertebrae. Rupture of the ligamentous capsule of the cricoarytenoid joint probably occurs in most cases. In a case which we encountered, both arytenoid cartilages were dislocated backward in a fall against the top rail of a fence.[1] In another case a similar double dislocation was caused

by explosion of an autotruck tire.[1] In a third case the cricothyroid and both cricoarytenoid joints were dislocated by the jerk of a rope in an attempt to commit suicide by hanging.[1]

Burns of the larynx may complicate deep burns of the neck; endolaryngeal burns may be produced by inhaled flame, hot smoke or caustic vapors.

SYMPTOMS

Any one or more of the following symptoms may be present in a case of injury involving the larynx: hemoptysis; blood-spattering cough; hematemesis; nausea; hoarseness or aphonia; dyspnea; stridorous, hissing, wheezing, bubbling, gurgling or rattling respiration. Local pain is usually moderate. There may be cyanosis; more often there is ashy gray pallor even if there is obstructive dyspnea.

Locally the skin of the neck immediately after injury may appear normal. There may be ecchymosis, swelling, tenderness, hematoma or subcutaneous emphysema. If the larynx is obstructed, there will be indrawing at the suprasternal notch. If there is an open wound of the neck, the larynx and trachea may be laid bare in the wound. If either has been opened, the blood will be bubbling, and it will be spattered by coughing. An opening into the pharynx reveals itself by a flow of thick glairy saliva, and when water is taken the wound is flooded; if there is any attempt to swallow food, it is projected out over the wound.

The general symptoms are the same as in injury elsewhere. The patient may be unconscious from loss of blood, from shock or from concurrent injury of the central nervous system. There is no special laryngeal shock as was once thought and taught in connection with surgical operations.

DIAGNOSIS

The symptoms may or may not be suggestive, but examination of the larynx is indicated in every case of injury to the head or the neck, *even in the absence of any laryngeal symptom.*

Methods.—All four methods of examination, by mirror, by direct laryngoscope, by palpating finger and by roentgen ray, are necessary for completeness. There may be circumstances under which all four of these methods cannot be used immediately; the attendant must then do the best he can, remembering always that the laryngeal examination is incomplete and that thorough examination by all four methods must be done as soon as circumstances permit. The diagnosis of obstructive laryngeal dyspnea can always be made objectively by the indrawing at the suprasternal notch, at the clavicles and in the intercostal spaces. When these signs are present in any case of trauma in or near the larynx, low tracheotomy should be done without waiting for completion of the delayed diagnosis.

One method, palpation, is always promptly available, and it is invaluable. When the skin is not destroyed and before swelling has obliterated landmarks, the thyroid and cricoid cartilages can be palpated in their entirety by trained fingers, and in traumatic conditions, as well as in disease, information otherwise unobtainable can be elicited with certainty.

A fracture may be felt as two edges where there should be continuity. This usually means that the perichondrium has been ruptured, but not always. Crepitation may be elicited as a sensation, rarely as a sound. It is readily distinguished from the crackling sensation of emphysema by its giving a sensation of displacement of the air bubbles under digital pressure. Emphysema and fracture may coexist. In case of multiple fracture producing fragmentation the flexibility where there should be relative rigidity is unmistakable to the trained touch. The roentgen ray is an invaluable diagnostic means and should be used as soon as possible in

every case of laryngeal injury, obvious or suspected. A large number of normal roentgenograms for comparison will be helpful in interpretation because of the variation in the normal shadows of laryngeal cartilages.

When there is an open wound of the neck involving the larynx, complete exploration is best deferred until it can be done under aseptic precautions except so far as the control of hemorrhage or the forestalling of asphyxia may necessitate.

Mirror appearances will depend on the character of the injury and the time elapsed. A crush may show a crowding of the tissues into the airway. Soon there will be mucosal ecchymosis or submucosal hematoma, giving a reddish purplish or violet color; sometimes this is accompanied by blood or blood-tinged secretions. Soon, also, there may be submucosal emphysema with pallor of the mucosa. These conditions—displaced tissues, blood, hematoma, emphysema—may cause asphyxia before the onset of inflammation.

The appearances of traumatic laryngitis may be those of reddish mucosal inflammation or whitish watery edema, or there may be a visible cut or an obvious laceration. If the trauma is entirely submucosal, there will be, after a few days, suppuration with development of a paler color at the site of impending breaking through of pus.

A gash from a sharp-cutting edge, such as we have seen a number of times from a backed razor and once from a cleaver, also from a butcher knife, at first is easily overlooked. Soon, however, the cut edges swell, evert and are covered with exuberant granulations, sometimes with exudate.

A penetrating spent bullet may be visible as a bluish spot under the mucosa. We have seen a perforating bullet track with an endolaryngeal wound of entrance on one side and one of exit on the other. These openings were associated with a track from one side of the neck to the other.

In a case of stab with an ice pick the appearances were similar. There was a wound on each side of the neck—over the central portion of the left ala of the thyroid cartilage and at the posterior edge of the right wing. The endolaryngeal wound on the left side was in the ventricle; that on the right side was anterior to the arytenoid cartilage. It seemed probable that the ice pick had entered on the left side of the neck and emerged on the right, but this could not be positively determined. Palpation developed fracture of the thyroid cartilage.

Differentiation of Anatomic from Indirect and from Psychic Trauma.—Anatomic trauma of the larynx sometimes must be differentiated from indirect trauma and psychic trauma.[4] Each of these two conditions may occur separately or in combination. Either or both may be associated with anatomic trauma, and a clear understanding of the differentiation is essential.

Indirect trauma is structural injury of the larynx caused by violent action of the laryngeal muscles. One common form is a hematoma on one or both vocal cords ending in organization and inflammatory tumor formation. Ordinarily this is due to vocal abuse, singing, shouting, cheering at baseball or football games, extreme exhortation and the like. When associated with direct laryngeal trauma, it is due to screaming or yelling for help or because of pain or fright. In children it may be caused by shrieking.

By psychic trauma of the larynx is meant impairment of vocal function without structural abnormality of the larynx, due to accidental or other form of psychic shock. Though it is a distinct morbid entity, it may start from the same initial cause as, or it may follow, anatomic laryngeal trauma. The most important points of differentiation are that the patient with psychic trauma believes he cannot talk, yet he has air coming through his larynx that could be molded into a good

4. Jackson and Jackson.[1] Jackson.[2a]

whispered voice by the molds of speech, and when he is asked to cough, the cords are seen to approximate and vibrate, thus making the cough phonatory. This is a totally different thing from malingering. The patient with psychic trauma has a genuine ailment curable by proper treatment [1]; the malingerer is intentionally shamming to escape duty or to perpetrate a fraud. Psychic trauma is often the result of a shock that is commonly described as being "knocked speechless" or "struck dumb." Normal persons quickly recover their voices; in unstable persons the disability is prolonged into a chronic ailment.

COMPLICATIONS

It has become a tradition to enumerate pneumonia as a complication of trauma, even surgical trauma, of the larynx. Bronchology has taught us, however, that in many instances the so-called pneumonia is really bronchial obstruction due to a thick, tenacious secretion of such high viscosity that the cilia and the cough reflex cannot expel it. Over and over again we have seen all the physical signs on which the diagnosis of pneumonia was based disappear after bronchoscopic aspiration. So often has this occurred that we feel skeptical of any diagnosis of pneumonia in such cases unless confirmed by diagnostic bronchoscopy.[5] Precisely the same may be said of edema of the lungs.

Mediastinal emphysema is a common complication. It may be the result of either of two different mechanisms, seldom of both combined. With the skin of the neck unbroken, subcutaneous cervical emphysema may (more or less aided by muscular pressure and thoracic suction) extend between the tissue layers into the mediastinum; or with a large open wound involving the suprasternal notch, the inspiratory intrathoracic negative pressure may suck in air whose escape is prevented in a valvular way. That is to say, the air is drawn in on inspiration, but exit is hindered by the prompt crowding of the tissues into the thoracic aperture. The result is that intrathoracic pressure progressively increases until an atmospheric degree of pressure is reached and the patient is asphyxiated because no more air can be drawn in, or until the patient dies of pressure effects on the heart, the circulation and the innervation.

PROPHYLAXIS

Discussion of prevention of traumatic accidents is not within the scope of this paper. Prevention of laryngeal stenosis and of total atresia, on the contrary, are two subjects of utmost clinical importance. At all times the surgeon should have in mind the bearing of every clinical phase and of every element of treatment on these two clinical conditions, both of which are preventable. It seems to us necessary for emphasis to recognize two conditions instead of one in order to promote prevention of total atresia, because so many patients have come to us with the latter condition, which could have been easily prevented. The history usually is that after a brief and unsuccessful attempt at decannulation the practitioner became discouraged and allowed the patient to drift along until the laryngeal airway was totally obliterated. This is a calamity far exceeding stenosis in seriousness. There are two reasons for this: 1. So long as any air passes through the larynx the patient has a good laryngeal voice, whereas with complete occlusion the patient must acquire a buccal voice, using a pocketing of air in the pharynx, the esophagus or the stomach. 2. The treatment for cure of total atresia requires as a first step the making of a perforation through the occluding tissue. This operation not only requires skill and care but involves some degree of risk, whereas any sort

5. Mitchell, H. E.: Treatment of Victims of Poison Gas, Arch. Otolaryng. 37:372 (March) 1943.

of lumen, no matter how small, can be enlarged and shaped by means of core molds with little or no risk of mortality.

For the prevention of total atresia a braided silk thread worn "endless" in the larynx is sufficient, but this is only a makeshift for use when, for any reason, core molds are not available. It is better to begin at once with progressively increasing sizes of core molds, with a view to obtaining a cure of stenosis rather than merely the prevention of total atresia.

TREATMENT

The general medical and surgical care require no mention here; the subjects coming under these heads (shock, indications for transfusion and so on) are fully covered in surgical textbooks. There is no special shock from laryngeal reflexes as was once thought. There is, however, in some cases psychic trauma; this has been discussed in the section headed "Diagnosis."

In case of wounds involving the airway the use of morphine should be limited because of its depressive effect on the respiratory center and its inhibitory effect on the cough reflex.[18]

The outstanding features of the care of laryngeal wounds are concerned with the problems of asphyxia, hemorrhage, laryngeal stenosis and the maintenance of the vital "lines of communications," namely, the air and food passages. These fundamental facts must be impressed on every one concerned in the care of a patient with injury of the larynx or its neighborhood.

Examination of the External Wound.—Tracks of bullets or other missiles with small entrance and exit wounds are usually best left unexplored until there are positive evidences of suppuration. Even when done under favorable conditions, exploration usually results in infection. On the other hand, in many cases the track heals in a remarkable way. When suppuration is profuse, cleansing and search for foreign bodies, shreds of clothing and dirt may be indicated. Such an exploration may well be preceded by a roentgen examination. Subsequently a bismuth subnitrate mixture may be used for delineation, but not for the first examination. The use of a bismuth compound is good treatment for the track when treatment is indicated, but that of barium sulfate is not.

If the laryngeal wall has been extensively cut through or carried away, exploration of the interior of the larynx through the external wound may be done by using small retractors or a nasal speculum; this must be done gently and carefully so as not to denude any remaining cartilage of its perichondrium. If, however, the traumatic opening into the larynx is insufficient to permit exploration, it is unwise to enlarge the opening, because any extension for this purpose is almost certain to lead to impairment of the nutrition of the remaining cartilage, and in the always infected traumatized tissues chondral necrosis with long-continued suppuration ending in cicatricial stenosis is likely to follow. It is better to examine the laryngeal interior with the laryngeal mirror and the direct laryngoscope. Dealing with the always infected traumatized tissues of the larynx is a different thing from aseptic surgical laryngofissure for cancer of the larynx.

Tracheotomy.—Tracheotomy is indicated in practically every trauma of the larynx. It may be deferred only if the patient is constantly under observation and an operator is promptly available. When indrawing at the suprasternal notch is definitely present, tracheotomy should be done without delay, to prevent risk of asphyxia. This is well understood, but there are other indications not generally realized.

In the absence of indrawing tracheotomy is indicated for: (*a*) hemorrhage; (*b*) cervical emphysema to forestall mediastinal emphysema; (*c*) mediastinal

emphysema; (*d*) relief of tracheobronchial compression of any kind; (*e*) aspiration of inspirated or flowing blood; (*f*) removal of clots or other foreign material if peroral bronchoscopy is unavailable; (*g*) insufflation of oxygen (plus carbon dioxide, 7 per cent); (*h*) relief of pulmonary complications by frequent catheter aspiration; (*i*) thoracic penetration or perforation in some cases.

Tracheotomy may be indicated for hemorrhage in the absence of dyspnea under circumstances parallel to those calling for the emergency use of the tourniquet in traumatic hemorrhage of the extremities when search for bleeding vessels in the wound is not practicable. Obviously an ordinary tourniquet cannot be used on the neck. A potentially life-saving substitute is firm compression with use of a properly placed tracheal cannula to maintain the airway.[1a]

Tracheotomy should always be done low. High tracheotomy is a relic of the days when surgeons stood in awe of the thyroid gland and is sure to cause difficult decannulation and chronic stenosis.[6] If the trauma has laid open the larynx or the trachea near the larynx it is permissible to insert a cannula in this high position only in case of extreme emergency and then only temporarily. At once or as soon as possible tracheotomy should be done low for the wearing of a cannula, even if there are prospects of only temporary need of the cannula. It is best also, when possible, to have a few uncut tracheal rings between the wound and the cannular incision.

Resuscitation After Tracheotomy.—When tracheotomy is done late, as it usually is, respiratory movements may have ceased. The best method of dealing with this condition is by bronchoscopic aspiration and bronchoscopic insufflation of oxygen (plus carbon dioxide, 7 per cent). If equipment for this is lacking, the Silvester method of artificial respiration is best in cases in which the tracheotomic wound needs attention, as for arrest of hemorrhage. If it does not need attention, the prone pressure method is best. The tracheotomy has eliminated the lingula death zone.[7]

First Aid Against Asphyxia Without Tracheotomy.—Apropos of the treatment for asphyxia as a first aid measure without tracheotomy, it seems necessary to emphasize the importance of eliminating the lingual death zone[7] before the first stroke of artificial respiration. Unquestionably many patients have been killed by the first stroke of artificial respiration, which forced the residual air out of the lungs while the check valve action of the tongue prevented ingress. This is especially true of the prone pressure method, the first stroke of which drives the air out of the lungs. With the tongue properly held forward, however, this is the best of all manual methods. Additionally, as shown by Yandel Henderson, it has the advantage of aiding the failing circulation by forcing blood toward the heart. When oxygen is available, it should have an admixture of carbon dioxide to a level of about 7 per cent, as advised by Henderson. Every civil ambulance and fire house should have a tank of this mixture. Pulmotors and resuscitators are not to be recommended. The Drinker apparatus is excellent but is seldom quickly available in emergencies.

Débridement.—In case of lacerated and contused external wounds in the neighborhood of the larynx, débridement should not be done unless good illumination is available, and even then it should be extremely conservative. Excision should be limited to obviously devitalized tissue. Almost any surviving tissue near

6. Jackson C.: High Tracheotomy and Other Errors the Chief Cause of Chronic Laryngeal Stenosis, Surg., Gynec. & Obst. **32**:392 (May) 1921. Jackson and Jackson.[1]

7. Jackson, C., and Jackson, C. L.: The Lingual Death Zone in Asphyxia, New York State J. Med. **34**:1 (Aug. 1) 1934.

the larynx may help in building a support for the anterior wall of the pharynx; even if this wall is intact at first, it may later be opened by sloughing. Muscular tissue makes good material for support; every vestige that has life in it should be retained. Every fragment of cartilage that is attached to viable perichondrium should be saved; on the other hand, a completely denuded or detached fragment of cartilage will certainly never become healed in, because the wound is not sterile. A fragment is certain to become a pyogenic sequestrum. The débridement and the prompt closure of wounds that have given such excellent results in modern surgery are not immediately applicable to wounds of the neck in which the laryngeal cartilages are shattered. A core mold should be placed and free drainage maintained with a gauze pack for a time.

Local Insufflation of Antiseptic Powder.—Local infective reactions call for more frequent removal of the core molds and a light dusting of the wound with an insufflator that will throw only a small amount of minutely divided dust. Heavy dusting from a dredge box is not suited to laryngeal applications of any powder. Before the days of the sulfonamide compounds we used insufflations of bismuth subnitrate. The subnitrate often turned dark, sometimes giving the wound an alarming color; but until the advent of the sulfonamide compounds there was nothing to equal the antiseptic power of the bismuth compound used lightly as a dusting powder by insufflation. Lumps of powder of any kind must be avoided in endolaryngeal applications.

In open wounds of the neck the now generally accepted local application of the sulfonamide compounds is not contraindicated, though it is well to cover openings into the air or food passages momentarily to prevent entrance of uncontrolled amounts of these drugs. Direct applications of limited amounts to wounded as well as infected mucosal surfaces are being used successfully, but obviously the amounts used must be controlled. There remains much to be determined as to both toxicity and sensitization in the use of the sulfonamide compounds, but now abundant clinical evidence warrants the local use of these powerful agents in the control of infection and contamination, even prior to bacteriologic studies in the particular case. Time may be required for the development of accurate chemotherapy based on determinations of the particular organisms present or predominating.

Treatment of Crush, Fracture and Dislocation by Means of Core Molds.—In a crush of the laryngeal box the cartilages may be bent, fractured or fragmented. The whole future usefulness of that larynx depends then on prompt and proper care. The best form of treatment is that employing core molds; they serve as internal splints on which the natural form of a laryngeal lumen is maintained or rebuilt. As a rule, the sooner the core mold is placed, the better will be the result. Early placement is especially imperative, because the deformity usually associated with a crush will soon become fixed, and many months may be required for restoration; whereas if a properly fitting core mold is put in place immediately, the structures are restored to their proper locations and become fixed there, thus establishing a good laryngeal framework. The core mold should not be as close a fit as is necessary to promote absorption in dealing with cicatricial conditions of long standing.

Simple fracture of the laryngeal cartilages, without fragmentation or crushing, and not compound, usually calls for immediate insertion of a core mold as an internal splint. It should not be of too large a size. If there is much endolaryngeal swelling, it should be a rather close fit in the narrowed lumen. Larger

sizes can be progressively substituted as the swelling subsides. This subsidence will be rapid as compared with that of cicatricial tissue in chronic stenosis, unless the fracture is compound; in this case there is always perichondritis with suppuration ending in chondral necrosis. Here again core molds are the best form of treatment, as mentioned in another paragraph.

Dislocation of one or both arytenoid cartilages can be reduced by the insertion and wearing of a core mold of large diameter if the patient is seen within a few days after the accident. For this, tracheotomy must be done if it has not already been performed for prevention of asphyxia. If the dislocation has been unrecognized until fixation in the dislocated position has occurred, reduction with restoration of good movement is impossible.

The selection of the proper time for beginning the core mold treatment is of utmost importance in trauma of the larynx. If there is a fracture of the thyroid or of the cricoid cartilage without any damage to the skin or the mucosa, a core mold of an easy fitting size may be inserted at once. If, however, there is trauma of the interior of the larynx, the first core mold should not be inserted until the virulence of the infection has been determined or until the necrotic tissue has sloughed off and the raw surfaces are covered with healthy granulations. This under treatment with a sulfonamide compound will ordinarily require only a week or two, unless the infection is exceptionally virulent. Epithelization should not be awaited before beginning the insertion of core molds; these are the best means of promoting the essential epithelization with epidermal epithelium. If the patient does not come under observation until after a longer period has elapsed and the building up of inflammatory tissue has begun to narrow the laryngeal lumen, the core mold treatment should be begun at once. If the patient comes at a still later period, the case may be one of cicatricial stenosis or atresia. The treatment of this condition is considered in the subsequent section.

Treatment of Cicatricial Stenosis by Means of Core Molds.—When the larynx has been allowed to heal without core molding after trauma one or the other of two stenotic conditions may be encountered: If the trauma has been slight and limited to endolaryngeal soft tissues, the contraction of the cicatricial tissue may give rise to a weblike obstruction. If, however, there was primary damage to the perichondrium or to the cartilage, there is usually, in these long-standing cases,

---

EXPLANATION OF PLATE I

Above: At the left is shown a schematic cross section of the normal larynx. *V B* identifies the ventricular band; *V C*, the vocal cords; *T*, the thyroid cartilage; *C*, the cricoid cartilage. At the right is shown a cross section of a larynx and trachea as affected by massive cicatricial hyperplasia. *A A* indicates the hyperplastic tissue obliterating the lumen of the larynx below the level of the ventricular floor. It was especially firm and obliterative through the cricoid region.

Below: At the left is shown a schematic cross section of the injured larynx with a core mold of slender diameter. The tissue *A A* was so firm and filled the cricoid ring so completely that it prompted a proposal to cut the cricoid cartilage. At the right is represented a core mold of large size in position after progressively increasing sizes of molds had caused absorption of the hyperplastic mass (*A A* at right above). Disastrous cutting of the cricoid cartilage was shown to be unnecessary.

[NOTE.—All our clinical experience has been with core molds ground to shape out of pure rubber cord made from natural rubber. Cautioning against the use of any of the various forms of synthetic rubber seems advisable; no one knows what the reaction of the laryngeal tissues to them might be. "Synthetic rubber" is a physical, not a chemical, imitation.]

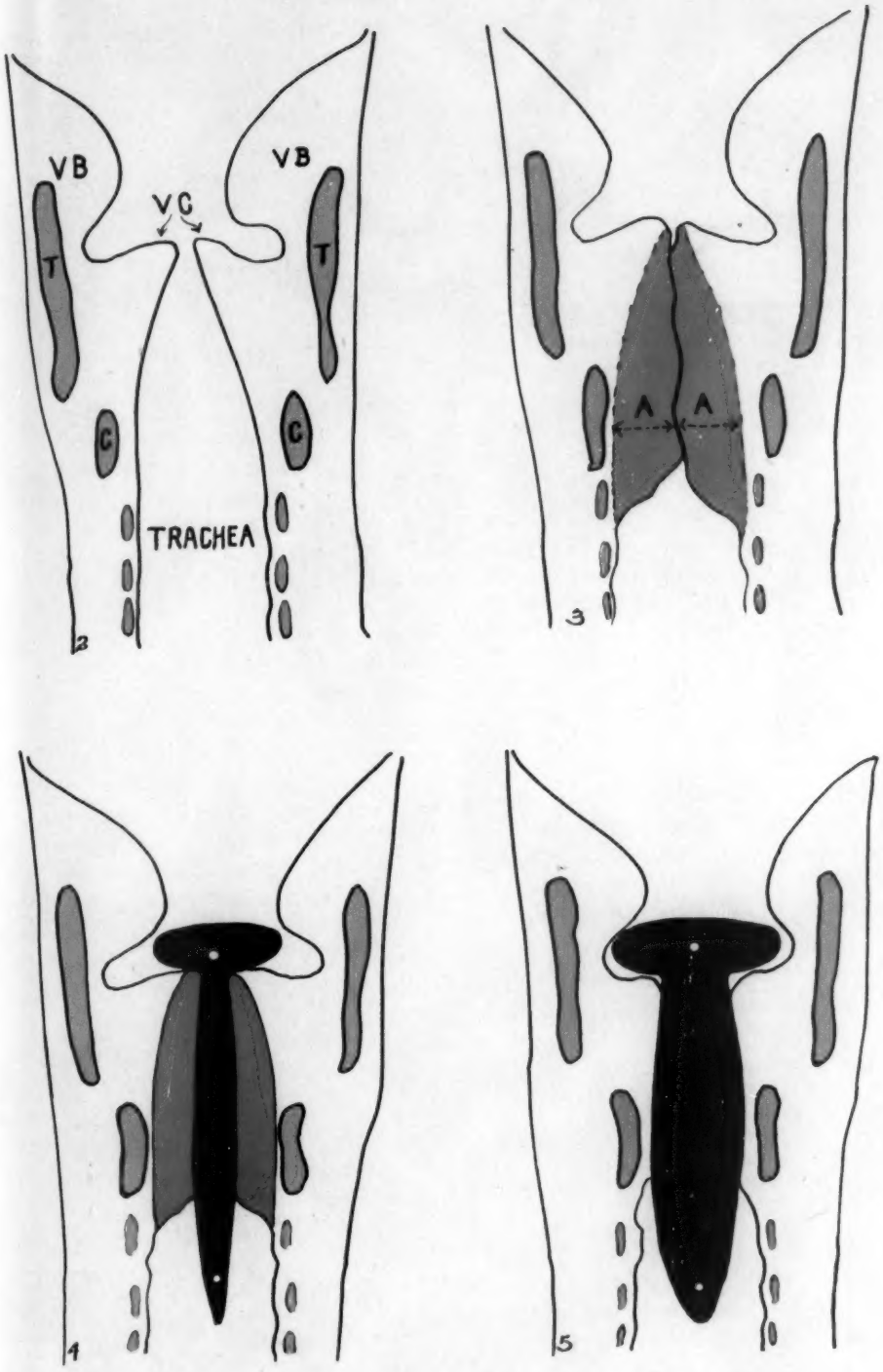


PLATE I

a mass of cicatricial tissue the contraction of which has nearly or quite obliterated the lumen.[8]

From the standpoint of treatment these chronic stenotic findings may be divided into two classes, namely, (1) those in which there is a passage way, however small it may be, and (2) those in which there is total obliteration of the lumen.

Any sort of lumen can be dilated with progressively larger sizes of core molds.[1] When there is total atresia, a perforation must be made. The technic of this has been given in full a number of times.[9] Tighter fitting sizes of core molds can be used in chronic stages of cicatricial stenosis; in acute stages the molds must not make much pressure.

One great advantage of core molds is that they are self-retaining by their shape; the supraglottic swell prevents sinking downward, the infraglottic swell prevents coughing out. Any apparatus requiring transfixion of the laryngeal cartilage for retention risks ruin of the cartilage by spreading perichondritis and invites pulmonary complications by long retention of a filthy apparatus.

The technic of the use of core molds has been given in detail elsewhere.[9] A few points require emphasis here. Of utmost importance is the selection of the proper core mold for the particular case. It must be long enough to reach below the narrowing. The lower end must be above the tracheotomic opening so that it can never occlude the opening. In diameter the core mold should not be a tight fit in an acute traumatic condition. In old cicatricial stenosis it should fit tightly enough to make the required degree of pressure, and as soon as absorption of the redundant cicatricial tissue makes a loose fit, another mold of slightly larger diameter should be substituted. In acute trauma the standard form should be used, because it is designed to reform the laryngeal lumen to a normal contour. There may be a chronic condition requiring a special shape, but as a rule it is better to use the standard form in progressively increasing diameters. Some ingeniously modified core molds for 2 special cases have been made by Dr. Charles Norris, who will report on them later.

Chronic cicatricial stenosis can be cured in almost all cases, but both patient and operator must be prepared for a long, painstaking task. In some cases the duration may be shortened somewhat by using Negus skin grafts.

Plastic Repair.—Loss of tissue may necessitate plastic repair. Soft tissue repairs collapse on inspiration. For stiffening it is necessary to transplant cartilage. The most satisfactory way is to implant cartilage of the desired size and shape in a selected adjacent area as a sterile procedure; after the cartilage has healed in, the pedunculated flap containing it is swung into place, forming a lid to the laryngeal trough which is thus propped open.[1]

Treatment When the Pharynx or the Esophagus Is Involved.—When the trauma involves not only the larynx but also the pharynx or the cervical portion of the esophagus, prompt and proper care is necessary to save life. Two measures are essential:

1. A soft rubber catheter of small diameter must be worn constantly through the nose, the distant end being in the thoracic portion of the esophagus. This tube serves four purposes. It (*a*) promotes esophageal drainage of secre-

8. Cummings, G. O.: Atresia of the Trachea Following Injury: Dilatation with Core Molds, Tr. Am. Broncho-Esophagological A., 1940, p. 38. Patterson, E. J.: Laryngeal Stenosis in Children, with Special Reference to Treatment with Core Molds, Arch. Otolaryng. **29**:71 (Jan.) 1939. Jackson, C.: Tracheobronchitis Due to Nitric Acid Fumes: Bronchoscopy, New York M. J. **104**:898 (Nov. 4) 1916. Jackson and Jackson.[1a] Imperatori.[3]

9. Jackson and Jackson.[1] Jackson.[2b]

tions alongside it, (*b*) enables maintenance of proper feeding and intake of fluids, (*c*) prevents dumping of food into the wound and (*d*) prevents total atresia of the pharynx and of the cervical portion of the esophagus. The last objective is a prime necessity, because any lumen, however small, can be safely dilated, whereas total atresia requires a perforating operation locally as well as gastrostomy externally.

2. There must be maintenance of a fully balanced diet and of an adequate intake of fluids. The total of a perfect formula must be taken each day; otherwise resistance will be lowered and healing will be retarded.

Plastic repair is necessary in many cases of extensive laryngeal and pharyngeal trauma. Good results are thus obtained, but each case is a particular problem.

Treatment of Endolaryngeal Burns.—Searing by inhaled flames, hot smoke or caustic vapors usually produces a thick, tenacious secretion from the laryngotracheobronchial mucosa. This secretion should be aspirated to prevent asphyxia. Apart from relieving bronchial obstruction, the clearing of the airway permits local medication. The best form of medication is a spray of a mild solution of sodium bicarbonate. Both aspiration and spraying can be done best with the direct laryngoscope or the bronchoscope. If these are not available, tracheotomy should be done, even if not required for dyspnea. It has the added advantage that the aspiration and the medication can both be done by the nurse, as often as necessary, every half-hour or oftener. For aspiration the nurse uses a soft rubber catheter.

PROGNOSIS

In the early stage of laryngeal injury the prognosis as to life depends almost entirely on the availability of surgical care. In the absence of tracheotomy asphyxiation occurs in almost all cases, from noninflammatory obstruction of the airway in most of them. When surgical care is available from the time of injury, few patients die from laryngeal causes.

In the later stages surgical care is almost always available, and the prognosis as to life is good.

When the best methods of treatment are available, the prognosis as to the airway is good in all cases in which there has been little or no loss of cartilage, though the duration of treatment may be long before permanent decannulation and fistular closure are obtained. Other things being equal, the shortest duration is observed in the cases in which the tracheotomy has been done low.

In regard to the food passage an involvement of the pharynx or of the cervical portion of the esophagus as well as of the larynx adds gravity to the immediate prognosis. If a lumen is maintained by wearing even the smallest catheter, a good swallowing tube can ultimately be obtained.

Prognosis as to the voice is similar to that for the airway. So long as air passes through the larynx the patient will have at least a good loud whispered voice. When the lumen becomes adequate, the voice will develop into a "stage

---

Explanation of Plate II

When the introduction of a slender core mold meets with much resistance, it may be assisted by traction from below. In order to make the traction axial, the push fork depresses the braided silk (*B*) into the trachea when traction is made. To prevent any possibility of plunging of the fork into the tissues, the operator grasps the push fork tightly and rests the ball of the thumb, *D D*, on the front of the patient's neck, *H*. As landmarks, the epiglottis, *E*, and the arytenoid (*A*) and cricoid (*C*) cartilages are represented. The hand is sketched ungloved for clearness.

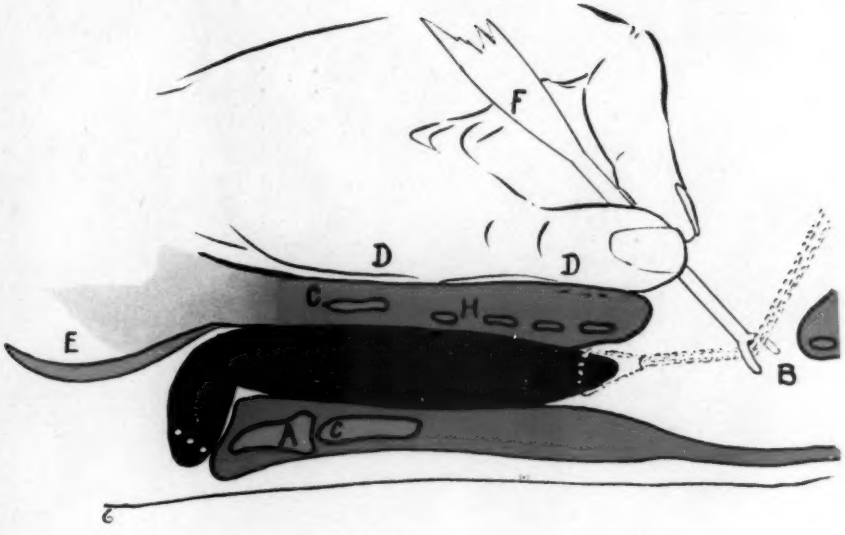


PLATE II

whisper"; later there will usually be loud phonation, rough in quality. In total atresia the voice is buccal, pharyngeal or esophageal until a small airway is restored. In cases of total destruction of the laryngeal framework, the airway cannot usually be restored; but the pseudovoice can be developed to a useful degree, just as after laryngectomy for cancer.[10]

SEQUELAE

Formerly, wearing a cannula for life was the usual sequel of severe and often of even slight laryngeal trauma. Core mold treatment has eliminated a large percentage of such sequelae.

Deformity requiring plastic repair apart from restoration of the airway, particularly the excision of an unsightly scar, occasionally is observed. Care must be taken in such work not to create any contractile tension that may lead to stenosis.

SUMMARY

Tracheotomy may be necessary for emergency control of cervical hemorrhage even in the absence of dyspnea. By enabling cannular maintenance of airway it permits compression in the neck to serve here as does the tourniquet in emergency control of hemorrhage in the extremities. Endolaryngeal hemorrhage can be readily controlled by tracheotomy followed by direct laryngoscopic packing of the larynx. Tracheotomy, done early and low, is indicated in almost all cases of endolaryngeal trauma.

In lacerated wounds of the neck involving the larynx the following points are important: (*a*) A cannula should not be worn in the wound more than a few hours if at all. To avoid this, low tracheotomy should be done at the earliest possible moment. (*b*) Débridement within the larynx is contraindicated. (*c*) The cervical wound should not be completely closed; a large channel down to the larynx should be packed open until perichondritis and chondral necrosis are controlled. (*d*) The laryngeal cartilage should not be stitched. (*e*) When the food passages are opened, a feeding catheter should be worn through the nose until the food passages are repaired by plastic or other methods; prevention of dehydration is even more important than feeding in these cases. (*f*) If sulfonamide compounds are to be used locally in cervical wounds with openings into the air or food passages, such openings should be momentarily closed with a pad of gauze to prevent entrance of excessive amounts of the drugs. Light and sparing dustings may be used on mucosal wounds.

In laryngeal fractures and crushes a core mold should be inserted immediately or within a few days to serve as an internal splint to maintain a good lumen and restore form.

The early use of such molds prevents chronic laryngeal stenosis. Usually their use can be commenced within a week or two after the occurrence of trauma.

Pneumonia and edema of the lungs are exceedingly rare complications. The signs on which such diagnoses have been based disappeared in most cases after bronchoscopic aspiration of obstructive secretions of high viscosity.

Psychic trauma with vocal manifestations is curable by psychic treatment in almost all cases.

255 South Seventeenth Street.

10. Jackson, C., and Jackson, C. L.: Cancer of the Larynx, Philadelphia, W. B. Saunders Company, 1939, p. 130. Jackson and Jackson.[1]

PROSTIGMINE THERAPY OF ATROPHIC RHINITIS

CAPTAIN ROBERT HENNER
MEDICAL CORPS, ARMY OF THE UNITED STATES
AND
WILLIAM BUSBY, M.D.
CHICAGO

Recent literature has pointed to new trends in the treatment of atrophic rhinitis. Soskin and Bernheimer [1] have pointed out that the good results reported with the use of estrogenic substances in the treatment of atrophic rhinitis were due to the secondary hyperemia associated with direct nasal application of estrogens. They expressed the belief that the primary hormonal effects were not the essential factor and used prostigmine methylsulfate successfully to produce a prolonged hyperemia in patients with atrophic rhinitis. Whether the pathologic change of atrophic rhinitis is primary, as the so-called ozena, or secondary in type, resulting from infection, the atrophy is dependent on limitation of the blood supply. The other changes, such as mucoid degeneration of the epithelium with later squamification, periglandular lymphocytic infiltration, glandular proliferation and diminution in volume of tissue, are directly and indirectly related to the local changes in blood volume. In ozena the obliterative endarteritis brings these changes, whereas in secondary atrophic rhinitis the inflammatory changes may bring about hyalinization of the walls of the blood vessels, with diminution of blood supply and resultant atrophy.

Having observed these pathologic changes, one of us (R. H.) in 1936 began the use of mecholyl in an attempt to stimulate blood supply. Because of the extensive side reactions associated with the use of this drug, such as salivation, tachycardia and flushing, and because the beneficial effects were fleeting, the method was discarded. However, in 1 case, that of a Mexican girl aged 12, whose entire family had typical primary ozena, excellent results were achieved. At the time it was felt that the lasting results were effected because of the relatively early stage of the disease and the reactivity of a child's tissues.

We felt that perhaps prostigmine, a parasympathetic stimulant with more prolonged effects, might give better results. The question always arose as to whether the treatment of atrophic rhinitis resulted in real improvement or merely in greater comfort through rigorous care. In order to measure objective improvement in patients with atrophic rhinitis we decided to study a series of cases under good control in which prostigmine methylsulfate therapy was employed and to use the nasal temperature as an objective measure for determining the effect of treatment. It was felt that with the widening of the nasal chambers present in atrophic rhinitis the temperature in typical cases would be substantially below the normal nasal temperature. We believed also that if therapy aimed at increasing blood supply was effective this would be reflected by an increase in the nasal temperature.

From the Illinois Eye and Ear Infirmary.

Ampules of prostigmine methylsulfate (1:2,000), 1 cc., were supplied for this study by Hoffmann-La Roche, Inc., Nutley, N. J.

1. Soskin, S., and Bernheimer, L. B.: Mechanism of Estrogen Effect on Nasal Mucosa in Atrophic Rhinitis: Successful Treatment with Prostigmin, Proc. Soc. Exper. Biol. & Med **42**:223, 1939.

METHOD

Twelve cases of atrophic rhinitis of varying types in patients of various ages were selected and studied clinically. Basal nasal temperatures were established and biopsies performed. The instrument used for studying the nasal temperature was the Taylor Dermatherm, which is a thermoelectric instrument for the rapid and accurate measurement of surface temperatures. The electrode was introduced into the nose, always on the same side, without cocainization, and the temperature reading (on a special millivoltmeter calibrated to read in degrees centigrade)

Weekly Averages of Temperature

Case	1	2	3	4	5	6	7	8
Side	Left	Right	Left	Right	Left	Left (advanced cardiac disease)	Right (sinusitis)	Right
Initial basal temp. (C.)	30.4	31.85	33.4	33.7	32.4	30.5	35.3	33.75
Week of Treatment 1	32.4	35	33.8	33.9 (Immediately after spray 34.3)	33.8	....	35.1	33.6
2	33.9 (Coryza)	34.5	33.6	.... (Coryza)	34.5	31.8	35.9	35.5
3	34	....	....	36.5	34	34.9	35.8	36
4	....	33.1	33.4	....	33.3	34	34.8	36.5
5	....	33.1	....	34.5	33.2	34.1	34.2	36.4
6	34.5	33.9	....	34.6	35.3	33.3	....	36.2
7	34	35	33.2	35	34.9	35.1	33.4	
8	33.9	34	35.5	35.5	33.4	35.3		
9	34.3	35.5	34.8	34.3	35.6 (Headache)	35.3	34.3	
10	35.7	35	35.4	35	35.6	33.6	35.4	
11	35.9	33.4 (Coryza)	No treatment	34.3	36.3 (Headache)	35.4		
12	35.3	35.7	No treatment; symptoms returned	35.2	....	Cardiac failure		
13	35.1	34.3	34.7	35.2	....	32.3		
14	....	35.1	35.7	36.4	....	Treatment discontinued		
15	35.2	35.2	34.9	36.1				
16	35.1	35.4	....	35.8				
17	35.8	34.5	....	36.0				
18	36.4	35.3	34.7	36.1				
19	36.2							

recorded. When the basal temperature was established by repeated readings, the patient was supplied with a solution of prostigmine methylsulfate (1:2,000) and was instructed to spray his nasal passages, using 0.25 cc. on each side, four times daily. All the patients were particularly instructed while using the prostigmine methylsulfate to continue with their usual treatments, such as irrigation with saline solutions, without variance as to the type or number of washes or the time of day.

OBSERVATIONS

The accompanying table summarizes the readings of nasal temperature during the course of therapy. Four of the cases were not reported because of discontinuation of therapy before a reasonable time had passed.

REPORT OF CASES

CASE 1.—L. C., a woman aged 34, complained of frontal headaches, nasal discomfort and excessive crusting, particularly on the left side. The diagnosis, corroborated by roentgenograms, was that of sinusitis with secondary atrophic rhinitis. Ozena was not present. Her basal nasal temperature on the left side was established at 30.4 C. (86.7 F.). The Wassermann reaction of the blood was negative. Biopsy revealed hypertrophied stratified squamous cell epithelium, normal basement membrane and moderate infiltration of cells showing chronic inflammatory changes in the stroma. The walls of the blood vessels were thickened and hyalinized. During the second week of prostigmine therapy the patient experienced frontal headaches and what she seemed to think was an infection of the upper respiratory tract. After this episode the patient noted a considerable decrease in purulent drainage and crust formation. After eight weeks of treatment with prostigmine the nasal discharge was watery. In the twelfth week slight headaches recurred for a few days and then disappeared.

Results.—At the time of writing, six months after the study ended, the patient's nasal mucosa appears normal. Only an occasional crust is seen, and since the twelfth week she has experienced no headache. In the nineteenth week readings of the nasal temperature averaged 36.2 C. (97.2 F.), a rise of 5.8 degrees C. (10.4 degrees F.). A biopsy after the nineteenth week revealed thinning of the squamous epithelium and hypertrophy of normal basement membrane. There was an increase in subepithelial stroma, with many dilated capillaries, and a resolution of the cellular infiltration. Prostigmine therapy was continued, although no further temperature readings were taken. From both the patient's statements and our observations, it seems that in this case there has been definite improvement.

CASE 2.—A. H., a woman aged 38, complained of crusting and ozena of twenty years' duration. Her basal nasal temperature averaged 31.85 C. (89.3 F.). The Wassermann reaction of the blood was negative. A biopsy performed prior to the beginning of treatment revealed stratified squamous epithelium and in protected areas normal pseudostratified ciliated columnar epithelium. The basement membrane was mildly thickened, and there was moderate cellular infiltration. The stratified areas showed papillary hypertrophy. Two weeks after beginning treatment the patient noticed improvement. At that time the odor disappeared, and it has never recurred. She continued to improve until the eleventh week, when acute coryza with frontal headaches developed. In the eighteenth week of treatment her nasal temperature was 35.3 C. (95.5 F.).

Results.—The mucosa appeared normal, with no crusting or odor. An increase of 3.45 degrees C. (6.2 degrees F.) in the nasal temperature occurred with treatment. A biopsy performed at the conclusion of studies of the temperature revealed more marked papillation and thickening of the basement membrane; infiltration of many more plasma cells and Russell bodies was seen than at the biopsy performed before treatment.

CASE 3.—N. V., a girl aged 18, gave a history of atrophic rhinitis present since childhood, with excessive crusting and odor. Her basal nasal temperature was 33.4 C. (92.1 F.). The Wassermann reaction of the blood was negative. Biopsy performed before the beginning of treatment revealed stratified squamous epithelium with slight hyperkeratosis. The basement membrane was thickened. The stroma had an excessive increase of glands with periglandular infiltration and marked hyalinization of the walls of the blood vessels. Prostigmine therapy was instituted, and after three weeks of treatment an improvement was noticed. The odor abated and crusting decreased. No treatment was used from the tenth to the twelfth week. During this time there was a return of crust formation but no odor.

Results.—The patient felt that she was much improved. A total gain in temperature of 1.3 degrees C. (2.3 degrees F). was noted. Treatment was discontinued in the fifteenth week. The mucosa appeared improved, and a biopsy of tissue taken after treatment showed marked stratification and papillary hypertrophy, an increase in subepithelial tissue with dilated capillaries and formation of new blood vessels.

CASE 4.—M. L., a woman aged 42, gave a history of atrophic rhinitis present since the age of 6 years. An antrum window operation had been performed on the left maxillary sinus in 1940. She complained of excessive crusting and odor. A diagnosis of primary atrophic rhinitis was made. Her basal nasal temperature at the beginning of therapy was 33.7 C. (92.6 F.). The Wassermann reaction of the blood was negative. A biopsy made at this time revealed stratified squamous epithelium with papillary hypertrophy. The basement membrane was thickened; there were mild infiltration about the glands and a moderate amount of fibrosis

of the stroma. One week after beginning treatment the patient complained of frontal headaches in addition to an infection of the upper respiratory tract. Treatment was continued, and during the third to the fourth week a definite improvement was noticed. There was no crusting, but a thin purulent discharge was still present. Another episode of coryza occurred in the seventh week. The patient voluntarily discontinued irrigation and any treatment other than the use of the prostigmine spray.

Results.—There was an apparent increase in thickness of the mucosa, with diminishing of the nasal spaces. The final temperature, in the eighteenth week, was 36.1 C. (97 F.), a rise of 3.4 degrees C. (6.1 degrees F.). Biopsy of tissue taken at this time revealed conditions similar to those seen at the first biopsy except that some of the protected areas showed pseudostratified ciliated columnar epithelium; the stroma showed an increase of connective tissue.

Case 5.—J. B., a man aged 28, gave a history of atrophic rhinitis of seven years' duration, with excessive odor and crusting and occasional hemorrhage. He worked in dust and fumes. He had been rejected by the Army because of ozena. His basal nasal temperature was established at 32.4 C. (90.3 F.). The Wassermann reaction of the blood was negative. A biopsy at the beginning of treatment showed complete stratification, normal condition of the basement membrane, periglandular infiltration and hyalinization of blood vessels. There were a moderate infiltration and fibrosis of the stroma. In the fourth week after therapy was begun, a definite improvement was shown; the odor was absent, and, although there was a mucoid discharge, the crusting had disappeared. The patient experienced frontal headaches during the ninth and tenth weeks of treatment. In the eleventh week he had a relapse following headaches, with a slight return of odor and an increase in crusting. After this relapse, however, he continued to improve. The use of the prostigmine spray was discontinued after the twenty-second week of treatment, and by the twenty-fifth week a relapse had begun. The nasal temperature in the eleventh week was 36.3 C. (97.3 F.), a rise of 3.9 degress C. (7 degrees F.). The final biopsy revealed more marked stratification, and dilatation of the subepithelial capillaries, which appeared more nearly normal. The cellular infiltration was apparently at a later stage, as evidenced by the presence of more plasma cells and fewer lymphocytes.

Case 6.—C. M., a man aged 64, had repeated attacks of cardiac failure. He gave a history of atropic rhinitis of five years' duration with excessive crusting but no odor. His nasal temperature was 30.5 C. (86.9 F.). The Wassermann reaction of the blood was negative. Biopsy at the beginning of treatment revealed an almost complete stratification, with a normal condition of the basement membrane, periglandular infilration and advanced hyalinization of the deeper blood vessels. During the fourth week of treatment an improvement was noted, in that there were less crusting and greater ease in breathing. In the ninth week the patient experienced frontal headaches. In the eleventh week the nasal temperature was 35.4 C. (95.7 F.), which represented a gain of 4.9 degrees C. (8.8 degrees F.). In the thirteenth week when the patient presented himself he had an acute cardiac decompensation with dyspnea and swelling of the ankles. His nasal temperature was 32.8 C. (91 F.). All nasal treatment was discontinued, and he was referred to an internist for general care.

Results.—Our impression is that the atrophic rhinitis was definitely diminished until cardiac decompensation began to develop.

Case 7.—O. A., a woman aged 38, gave a history of having had atrophic rhinitis with excessive crusting and odor and loss of sense of smell since childhood. She had had previous intranasal surgical treatment. Her basal nasal temperature was established at 35.3 C. (95.5 F.). The Wassermann reaction of the blood was negative. The biopsy at beginning of therapy revealed marked stratification, but hyperkeratosis of the squamous cell epithelium was not observed. The basement membrane was thickened. There were excessive mucous glands and marked hyalinization of the walls of the blood vessels, with excessive fibrosis. In the first week of treatment there was a decrease in crusting and odor, and in the third week the patient's sense of smell returned. In the fourth week her nasal temperature was 34.8 C. (94.6 F.). At this time an acute coryza developed and her condition became slightly worse. Her final nasal temperature reading, taken in the ninth week of treatment, was 35.4 C. (95.7 F.). She is continuing the treatment and has had continued relapses every time she has stopped the use of the prostigmine spray for a few weeks. The final biopsy, performed in the tenth week, showed no significant changes.

Results.—Clinically, improvement has been demonstrated by disappearance of odor and return of sense of smell. However, in this case the characteristically low basal nasal temperature was absent and the rise after therapy was negligible.

CASE 8.—M. P., a woman aged 51, gave a history of atrophic rhinnitis of twenty years' duration, with marked crusting and ozena. Her basal nasal temperature was 33.75 C. (92.3 F.). The Wassermann reaction of the blood was negative. A biopsy performed before treatment was begun revealed stratification of the squamous epithelium with slight hyperkeratosis. The basement membrane was thickened. There were excessive mucous glands with marked periglandular infiltration and hyalinization of the walls of the blood vessels. During the first week of treatment the ozena disappeared and there was a decrease in crusting. In the sixth week the patient experienced frontal headaches lasting for two weeks. She began to improve in the eighth week and has since had no ozena and only slight crusting. Her nasal temperature rose to 36.5 C. (97.7 F.), a gain of 3 degrees C. (5.4 degrees F.). Temperature readings were discontinued in the seventh week, but therapy was continued.

Results.—In the twentieth week a biopsy showed slightly thinner stratified squamous epithelium; the basement membrane showed no thickening. There was a marked increase in subepithelial stroma, with many dilated capillaries, but the deeper vessels were unchanged. At the time of writing, six months after studies of the temperature were discontinued, odor is no longer present and crusting is reduced to a minimal degree.

Photomicrograph (case 3) of subepithelial area showing newly formed dilated capillaries (×104).

PATHOLOGIC OBSERVATIONS

Biopsies were performed prior to beginning therapy and on completion of a reasonable period of treatment. It was hoped that certain definite pathologic observations might be made, either in regard to resolution of the infection or, more particularly, in regard to changes in the blood vessels. It must be remembered that examination of small specimens of nasal tissue is a highly unsatisfactory basis for positive statements concerning pathologic changes which occur under therapy, because areas differ and cyclic changes occur. Therefore, a study of this kind cannot be controlled well enough to permit positive interpretations.

However, the following pathologic changes were observed to be typical in the cases studied: The superficial epithelium had undergone metaplasia to stratified squamous epithelium. The free surface was often papillated and sometimes hyper-

keratotic. A few protected areas retained pseudostratified ciliated columnar epithelium. The cylindric cells present were usually undergoing mucoid degeneration. The basement membrane was usually moderately thickened. Subepithelially, the principal change was an increase in mucous glands with periglandular lymphocytic infiltration plus the presence of some plasma cells and occasional Russell bodies. The walls of the deeper vessels were often greatly thickened and hyalinized and occasionally showed evidence of obliterative changes.

If any statement can be made on the basis of the biopsies performed after treatment, it seems that the following changes should be mentioned:

1. Thinning of the epithelium and a tendency to resolution of the inflammatory changes occurred. Fewer lymphocytes were present, and a comparative increase in the number of plasma cells was noted, as well as a deposition of new fibrocytes.

2. An increase of the subepithelial area with many new capillaries was seen. The capillaries were dilated and filled with red cells. The endothelium was hyperplastic, and mitotic figures were seen.

3. Fewer glands were observed. No effect was observed on the deeper vessels.

We wish to stress that these pathologic observations are not final and that further observation should be made; however, the increased blood supply by newly formed capillaries in the subepithelial areas may be significant.

SUMMARY

Eight well controlled cases of atrophic rhinitis were studied. The patients were under treatment with prostigmine methylsulfate (1:2,000) administered as a nasal spray. Objective results were studied by before and after biopsies and weekly readings of the nasal temperature taken with a thermoelectric instrument. An attempt was made to determine from these objective observations, plus the subjective reports of the patients, whether or not atrophic rhinitis is diminished under prostigmine methylsulfate therapy. The readings of the nasal temperature were used as an indicator of value of therapy on the supposition that a nasal spray of prostigmine can be effective through its parasympathetic stimulatory action, resulting in an increase of the blood supply to the atrophic areas and thereby effecting a revitalization of the atrophic membranes. Mechanically this should be reflected in narrowing of the widened nasal chambers.

During the course of treatment patients occasionally complained of periods of several days during which frontal headaches occurred, and subjectively they felt worse. Definite nasal changes were not observed at these times. However, the patients often said that they felt as if they had colds. It was felt that these symptoms were related to vascular effects of the treatment. However, because of the minimal effects of these symptoms, therapy was continued without any untoward effects and eventuated in progressive improvement.

One case of atrophic rhinitis secondary to sinus disease is reported. In this case the basal temperatures were never typically low and only a slight rise in temperature occurred under therapy. Although the patient experienced a return of her sense of smell, disappearance of ozena and a definite decrease of crusting, no microscopic improvements were demonstrated.

In all the cases here reported clinical therapy has been continued, although the temperature readings have been discontinued.

CONCLUSIONS

1. Atrophic nasal membranes have lowered temperatures.

2. Treatment with prostigmine methylsulfate administered as a nasal spray alleviates the symptoms of atrophic rhinitis. (Subjectively, all the patients claimed improvement.)

3. Rises in nasal temperature of as much as 5.8 degrees C. (6.4 degrees F.) occur with prostigmine therapy. Odor disappears in from one to four weeks and never recurs even in relapses as long as therapy is continued.

4. Crusting is definitely diminished and in some cases abates altogether. Patients in whom the disease is far advanced must be treated for about a year before an attempt can be made to interrupt treatment. Patients who are treated when the disease is in an early stage are sometimes improved promptly.

PATHWAY OF THE TONSILLAR LYMPHOCYTE

GEORGE KELEMEN, M.D.
BOSTON

In embryonic life, as soon as lymph cells accumulate around the crypts, which are hollowed out from endodermal buds of the second branchial pouch as a primordium of the tonsil, they penetrate into the lumens of the crypts, passing through the epithelium, with reticulization of the latter. This manifestation of lymphocytic activity continues until the last vestige of the tonsil disappears, on completion of its involution. This transfer of lymphocytes commences before the lymphoid tissue around the crypts differentiates into secondary nodules, and the process continues throughout the period of regression, after the nodules have disintegrated, only residual accumulations of lymphocytes being left around the remnants of the crypts. Many phases of this activity still remain controversial questions. For example, in the parenchyma, the clear centers of the secondary nodules represent "germinal" tissue according to Flemming,[1] whereas Hellman[2] expressed the belief that they are "activity" centers where lymphocytes are destroyed. In the epithelium, some observers believe that they can follow the various white blood cells, including lymphocytes, in their course through all the epithelial strata, whereas others think that these cells are destroyed within the layers of the epithelium. Also, some investigators hold that the penetration of lymphocytes takes place only in the deep portions of the crypts, whereas others believe it occurs throughout their entire extent. Moreover, some are of the opinion that once the lymphocytes have reached the lumen of a crypt they die, whereas others assert that they are conveyed along the lumen of the crypt to the surface of the tonsil, where they become transformed into salivary corpuscles.

The aim of the present paper is to offer a brief account of the successive steps in the life history and ultimate fate of the tonsillar lymphocyte. My own observations, both previously published and recent, are correlated with known facts, as established by other workers, in order to arrive at as fully integrated a picture as possible.

MATERIAL AND METHODS

In the present study, human tonsils which were removed at operation were fixed in a 10 per cent concentration of solution of formaldehyde U. S. P. Subsequently, they were embedded in paraffin and sectioned. The sections were prepared by three methods: (*a*) hematoxylin and eosin stain; (*b*) Maximow's azur II stain, and (*c*) Gömöri's method of impregnation with ammoniacal silver hydroxide.

The reticular framework of the diffuse tonsillar parenchyma was demonstrated in three ways. In previous investigations, Kelemen and Hasskó,[3] instead of using the technic of

This study was aided by a grant from the Charlton Fund.

From the Department of Otolaryngology, Tufts College Medical School.

Acknowledgment is made to Prof. LeRoy A. Schall, Harvard Medical School, for permission to use the laboratories of the Massachusetts Eye and Ear Infirmary.

1. Flemming, W.: Studien über Regeneration der Gewebe, Arch. f. mikr. Anat. **24**: 50-91 (Sept.) 1884.

2. Hellmann, S.: Der lymphatische Rachenring, in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1926, vol. 1, pp. 245-289.

3. Kelemen, G., and Hasskó, A.: Explantationsversuche mit Tonsillengewebe: II. Darstellung des Tonsillenreticulums in der Gewebekultur, Acta oto-laryng. **14**:101-112, 1930; III. Ueber Lymphocytenwanderung in der Gewebekultur bei Spontaninfektionen, ibid. **14**:112-123, 1930.

brushing or digestion of tissue, employed the method of explantation. After the free cellular elements have emigrated the reticulum is unmasked. In a further study, elective vital staining of the reticular fibers with neoarsphenamine, according to the method of Jancsó (Kelemen and Hasskó [4]) demonstrated clearly two forms of fibers in the reticulum: (1) a meshwork of coarse fibers, which leave the areas of the secondary nodules completely free, and (2) a system of much finer fibrils of tubular character, which are heavily laden with neoarsphenamine. These two systems of fibers represent merely two phases of development, as Maximow [5] claimed. He found in tissue cultures that when the wavy parallel fibrils of the argyrophil netwook increased in thickness, they gradually lost the ability to be impregnated with silver. Conversely, they began to stain by the methods used for collagenous fibers. Finally, in the present investigation, sections were studied after impregnation with ammoniacal silver hydroxide, according to the method of Gömöri.

According to Maximow, lymphoid tissue in general develops from mesenchymal cells, part of which differentiate into lymphocytes, whereas another part remain fixed, furnishing the reticular framework. Free and fixed cells remain closely associated in the reticular framework, a circumstance which was utilized in the present investigation to trace the mobile cells.

OBSERVATIONS

Like the neoarsphenamine technic (figures 2, 5 and 6, Kelemen and Hasskó [4]), Gömöri's impregnation method reveals that the clear centers of the secondary nodules are practically free from reticular fibers (fig. 1 *A*). The origin from the vessel walls of the fibers in the diffuse reticular meshwork is demonstrated in figure 2 *A* and *B*. These two fields contain at one corner a portion of a secondary nodule, free of fibers. The cells appear here merely as shadows within the reticular meshes.

The lymphocytes accumulate within the clear center of the secondary nodules in irregular rows or cords of cells, which are oriented with the long axis of the oval nodules. In the round cell accumulation of the dark caps, sparse fibers show up between the cells forming the first association of the two elements (fig. 1 *D*). Since the mesh of fibers is no looser here than in any other part of the parenchyma when observed by means of elective staining of the reticulum (fig. 1 *A*), it may merely be that the fibers are covered here by the denser cell groups.

The intimate coexistence of cells and fibers is seen everywhere in the diffuse lymphatic tissue, where the lymphocytes, after leaving the dark zones, form wedge-shaped groups before penetrating the epithelium, with the wedge directed against the latter. These formations were demonstrated clearly in tissue cultures of the migrating lymphocytes (Kelemen and Hasskó [3]).

Beginning at the boundary of the secondary nodule, one can trace the path followed by the lymphocytes with greater clarity by observing the attendant fibrils than by following the cells themselves, as the latter are often indistinct among other cellular elements.

The reticulum, as shown in figure 5 *A*, sharply outlines the border between the lymphatic parenchyma and the epithelium; the latter, however, as shown in figure 3 *A*, forms its own basal membrane, which must be ruptured by the penetrating elements.

Through this basal membrane and into the epithelium the reticulum sends projections (fig. 1 *B*), which envelop the outwandering group of cells like a fibrillary sheath. These projecting outshoots are sometimes seen in cross sections in ring form, without their connection to the main body of the parenchyma being

4. Kelemen, G., and Hasskó, A.: Ueber Fremdstoffaufnahme in den Tonsillen: I. Versuche mit Salvarsan an menschlichen Gaumenmandeln, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **130**:318-334 (Feb.) 1932.

5. Maximow, A. A.: Morphology of Mesenchymal Reactions, Arch. Path. **4**:557-606 (Oct.) 1927.

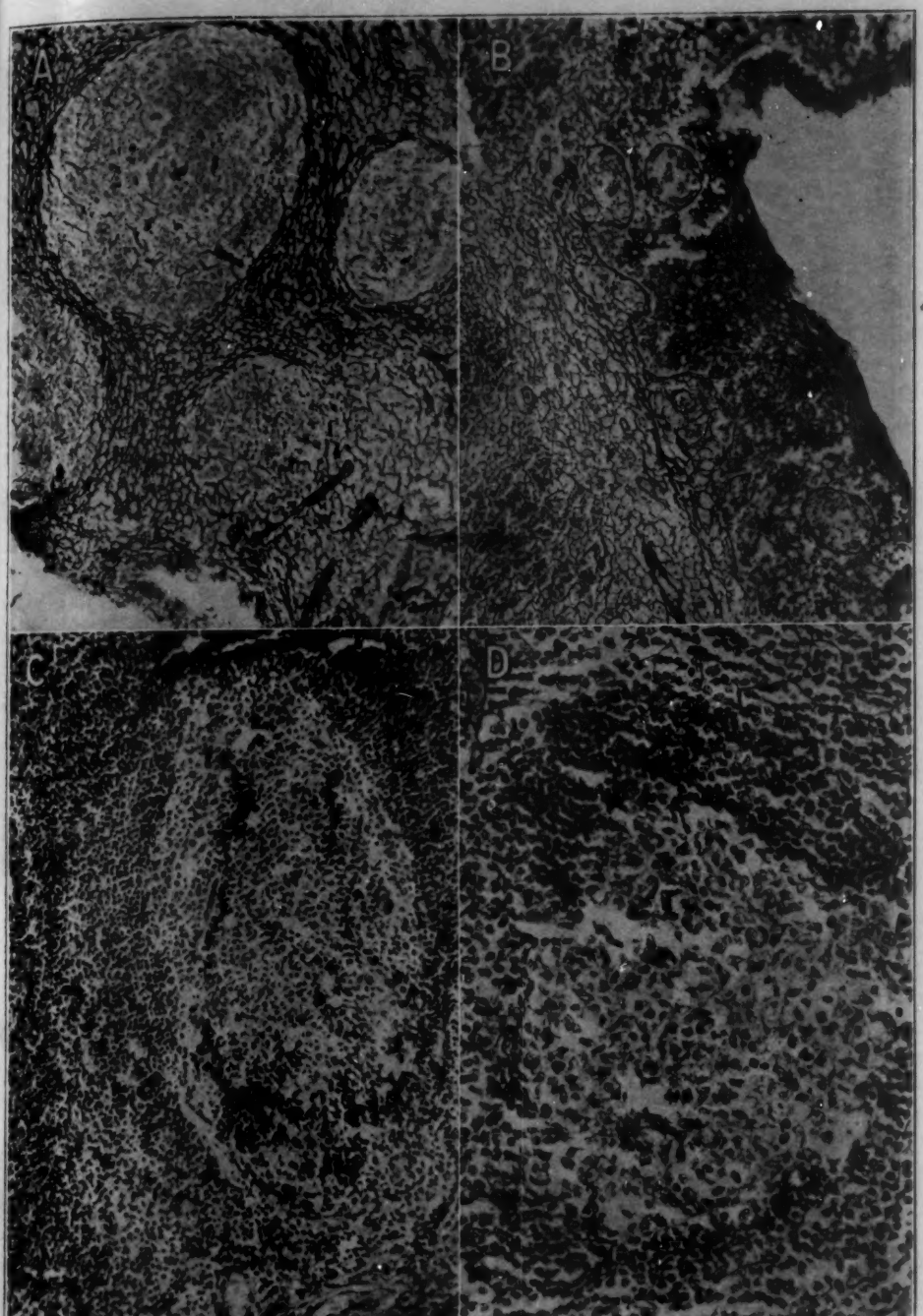


Fig. 1.—*A*, fibrillary meshwork of the basal reticulum, clearly outlining the area of the germinative centers. Gömöri impregnation.

B, penetration of reticular meshwork into the epithelium, with cross sections of penetrating projections, seen as round structures. Gömöri impregnation.

C, rows of lymphocytes within the light germinative center. Verhoeff-Van Gieson stain.

D, boundary of the dark zones, with sparse reticular fibers beginning to appear between the accumulated lymphocytes. Gömöri impregnation.

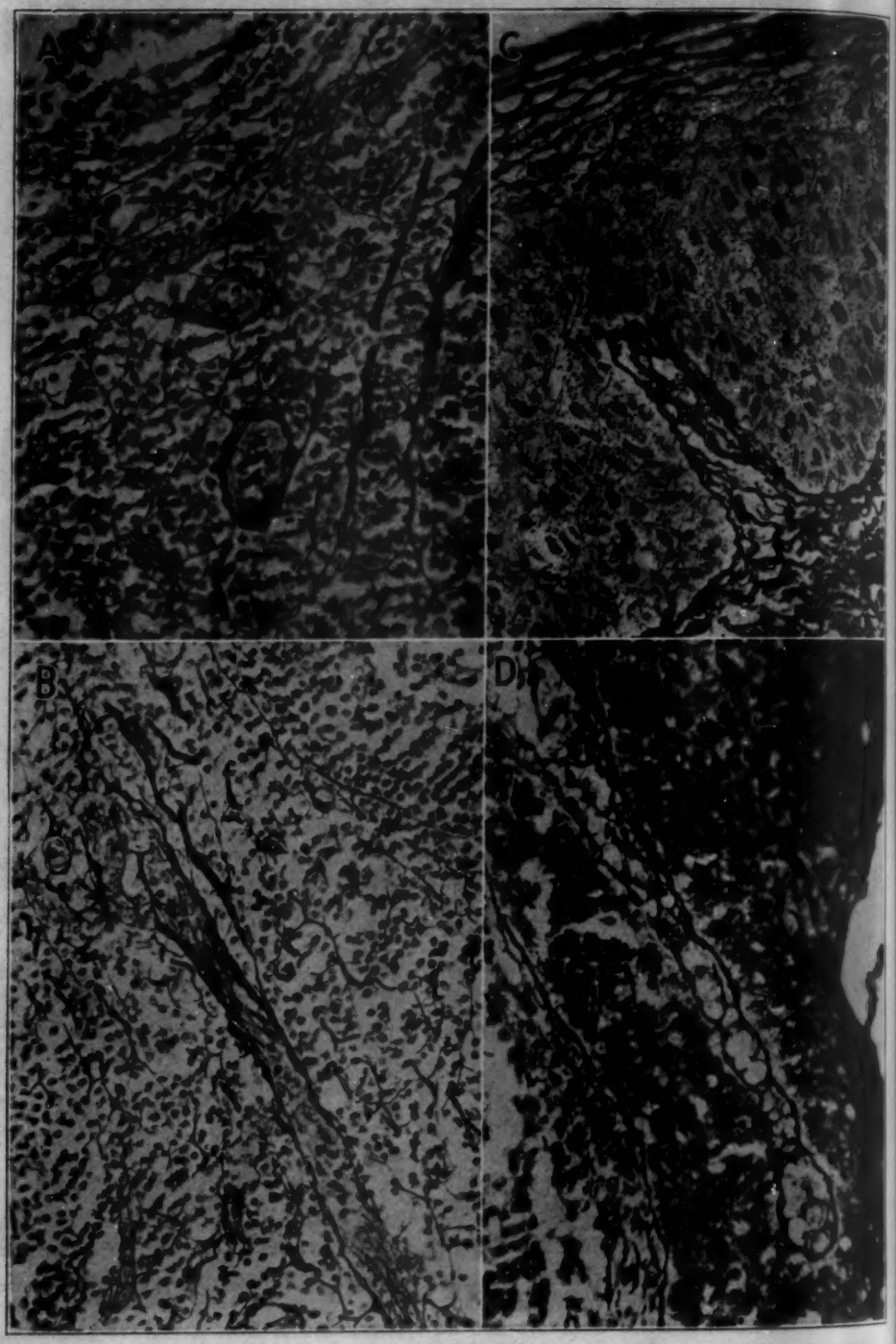


Figure 2

(See legend on opposite page)

apparent. They penetrate outward to touch the most superficial layer of the epithelial cells. Figure 2 *D* shows such a projection drilling its way through the epithelium, parallel to the surface, this course being taken possibly to evade a particularly strongly keratinized part of the latter. Figure 2 *C* shows a projection, in shape not unlike an ampullar crista, in screwlike action, piercing the epithelium. The surface is nearly reached by the spherical structure, as seen in figure 3 *B*.

Two sets of photomicrographs are presented to show the greater ease with which the process of transmigration is demonstrated if the fibrillary, rather than the cellular, elements are traced. Figure 4 *A* (hematoxylin and eosin stain) is taken from the depth of a crypt. In the corner, the epithelium is completely reticulized between two intact portions. The lymphocytes have reached the cryptal lumen and are swarming around the inclusion body in the lumen. *B* (fig. 4), a Gömöri impregnation, is comparable to *A* of the same figure. The process of penetration stands out with much greater clarity, as evidenced by the fibrous projections which can be seen entering the epithelium. In another set of illustrations, with higher magnification, three projecting cell groups are seen as stained with Maximow's azur II (fig. 3, *C*), while in an adjacent section (fig. 3 *D*) their respective fibrillary sheaths are demonstrated by silver impregnation. It is obvious that by the dual observations a much better survey and a more detailed picture of the process can be obtained.

The fibrous cage which envelops the lymphocytes does not penetrate farther than just beneath the superficial condensed layers of the epithelium; from this point the lymphocytes lose the protection of this cover.

That these cone-shaped, protective projections hitherto have escaped attention may be explained by the fact that the fibrillary network was always explored only within the strict limits of the diffuse lymphoid tissue of the parenchyma. These projections are quite different from the simple expansions of the diffuse lymphoid tissue between two papillae of the epithelium, as seen in figure 5 *A*. The few epithelial papillae clearly show all the characteristic layers of the epithelium and are penetrated by the projections in the same way as any other part of the epithelium, with or without papillae.

In figure 3 *A* the basal membrane is discontinued at two points. The penetrating cells are grouped within cystlike spaces. For better understanding of this disposition one may turn once more to the conditions as seen in the fibrillary network. The projections are more slender in the deeper half of the epithelium (fig. 2 *C*) and are thus more adapted to rapid penetration. As the projection approaches the surface, it seems that resistance is encountered from the condensed superficial cell layers, and the projecting structure spreads out. The result is a spherical, or cystlike, formation. The superficial epithelial cells are forced apart, as seen in figure 3 *A*, and the mobile elements are carried into the lumen of the crypt.

EXPLANATION OF FIGURE 2

A, concentric disposition of the reticular fibers, arising from vessel walls around the secondary nodule, seen at one corner of the section. Gömöri impregnation.

B, origin of reticular fibers from the vessel walls. In the meshes are shadows of lymphocytes. In one corner is a secondary nodule, which is free from fibrils. Gömöri impregnation.

C, fibrillary projection, drilling its way perpendicularly through the epithelium. Gömöri impregnation.

D, protruding fibrillary projection, running parallel with the cryptal surface. A secondary nodule, free from fibers, almost reaches the basal layers of epithelial cells. Gömöri impregnation.

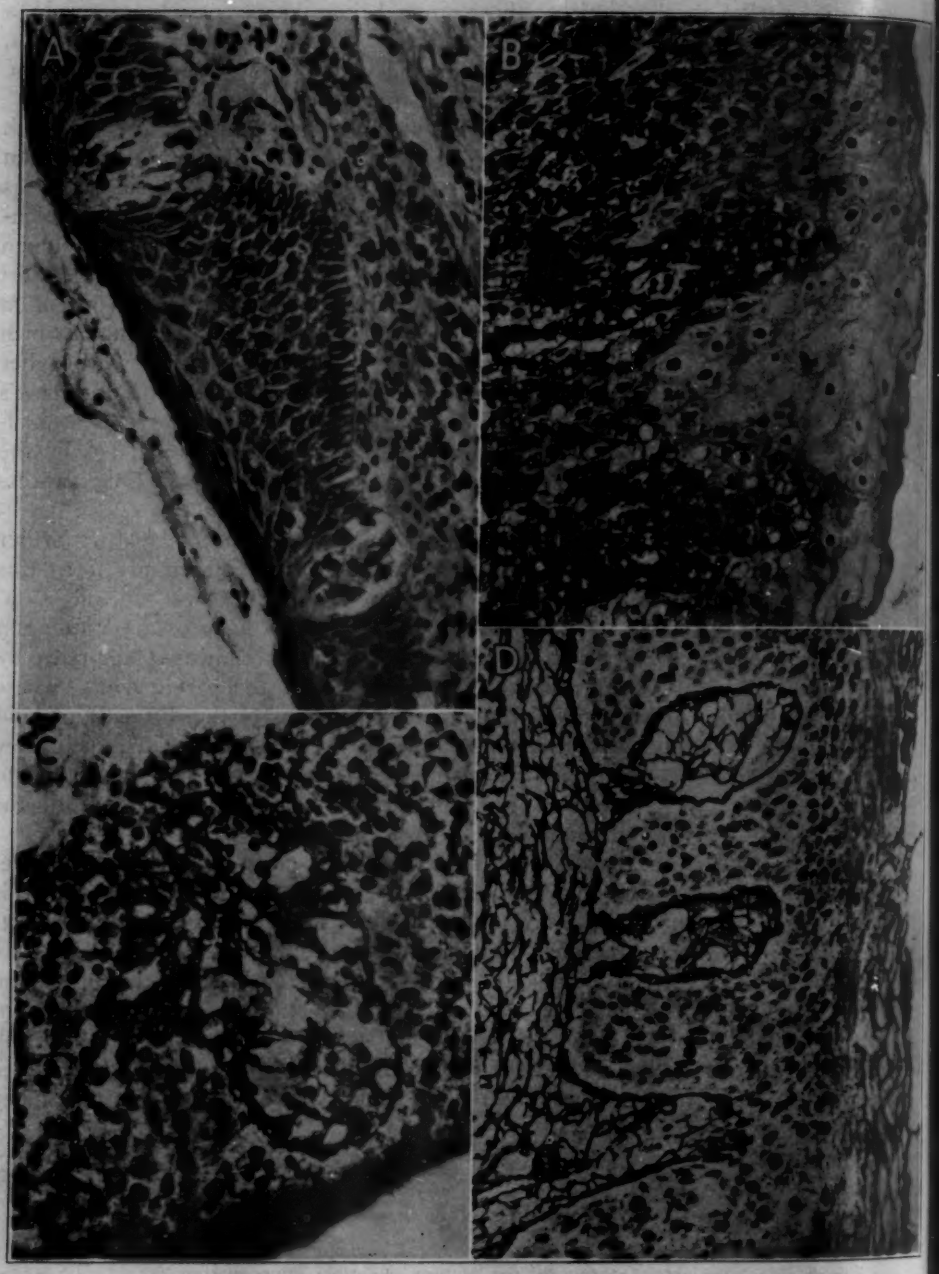


Fig. 3.—*A*, two cystlike structures extending the entire depth of the epithelium, which is about to rupture at its surface; basilar membrane at the base of the epithelium; transmigrated cells entangled in the fibrin mesh of the cryptal lumen. Hematoxylin-eosin stain.

B, fibrillary projection reaching the uppermost layers of epithelial cells, enlarging to a cystlike structure. Gömöri impregnation.

C, three transmigrating cell groups. Maximow's azur II stain.

D, three fibrillary projections, comparable to the projection in *B*. Gömöri impregnation.

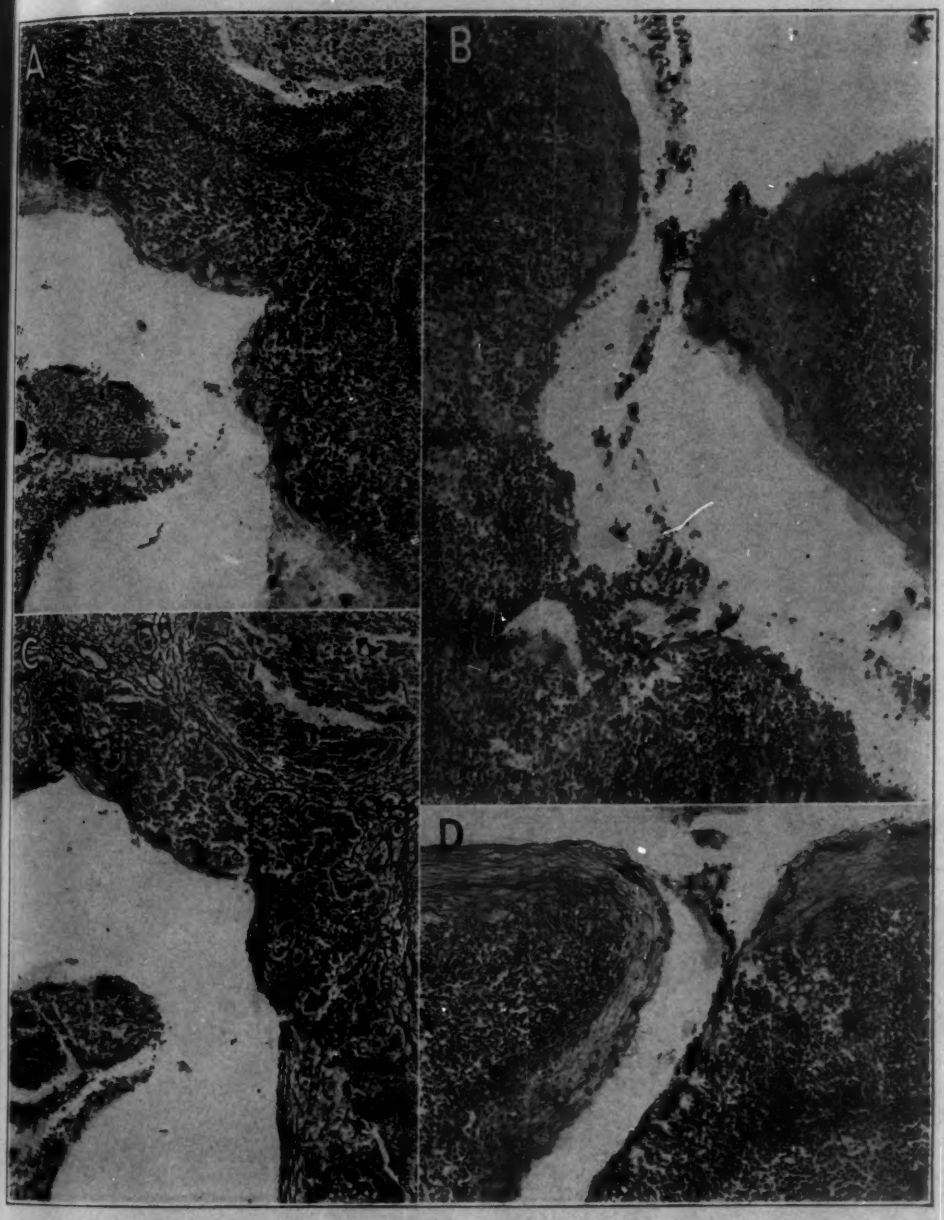


Fig. 4.—*A*, disposition at the bottom of a crypt: The epithelium is partly intact; one part is totally reticulized. A sharp boundary appears between the two areas. Lymphocytes, which have wandered through the epithelium, are gathered around a cryptal inclusion. Hematoxylin-eosin stain.

B, area comparable to that in *A*. The mechanism of reticulization of the epithelium is clearly visible, with manifold penetrating projections of all shapes; on their way outward, they have reached various levels. Gömöri impregnation.

C, rupture of epithelium at the bottom of a crypt. Transmigrated cells are moving toward the mouth of the crypt. Hematoxylin-eosin stain.

D, reticulization and rupture of the epithelium in the immediate vicinity of the cryptal orifice, with migrated cells entangled in the plug of mucus and fibrin at the mouth. Hematoxylin-eosin stain.

Fig. 5.—*A*, disposition of fibers between the secondary nodule and the epithelium, the latter being partly reticulized. A "papilla" of the epithelium penetrates deep into the diffuse lymphoid tissue. Gömöri impregnation.

B, orifice of a crypt. The fibrinous plug is composed of threads in the form of a tuft. Hematoxylin-eosin stain.

C, part of the section shown in *D*, at higher magnification, showing a tuft of fibrin threads.

D, reticulization of the epithelium of the crypt, which almost reaches the free surface. A tuft formation of fibrin appears at the cryptal orifice. Hematoxylin-eosin stain.

The further course of the lymphocytes can be traced in figure 3 *A*, in which the loss of fibrin and mucus in the crypts and at the free surface was prevented by omitting to wash the tonsil under running water, as is usually done before fixation.

It is claimed sometimes that the transmigration occurs only at the depth of the crypts and that the cells are then carried along the fibrin mesh to the orifice, as shown in figure 4 *C*. But this can happen also in the immediate vicinity of the orifice, as demonstrated in figure 4 *D*.

The lymphocytes, referred to from now on as salivary corpuscles, disappear from the crypts by a hitherto unnoticed disposition. The occluding "plug," which is composed of fibrin and mucus, can be differentiated into a tuftlike structure with endings resembling fimbriae. The latter carry the corpuscles onto the free surface. *B* and *D* figure 5 show this disposition; *C* is a portion of *D* photographed under higher magnification. The last of the lymphocytes are seen as they are caught in the layer of mucus (fig. 3 *A*) covering the surface of the tonsils.

COMMENT

From the abundant data in the literature concerning the histology and physiology of the tonsils, a few will be cited as having some bearing on the present observations.

Foot [6] emphasized that the fibers of the reticulum originate from the walls of the anastomosing blood vessels. Maximow [5] showed in tissue cultures of the spleen that the fibers of the reticulum are in direct contact with the adventitia of the vessels. The observations of Hasskó and myself are in complete accord with those of both these authors.

The disposition of the reticulum in relation to the secondary nodule has been discussed by several investigators. Hoepke [7] described a capsule around the nodule, which is ruptured frequently, the rupture permitting lymphocytes and reticulum cells to escape and being effected by an increase of pressure of lymph within the nodule; if the perinodal capsule does not rupture, the cells are pressed through the meshes and are transformed into "spears"; they may be forced in this form even through the epithelium, to be seen later in the crypts. Goodale [8] interpreted the follicle as undergoing rupture and compared it to a signet ring, the seal being directed always toward the lumen of the nearest crypt. Novak,[9] who used pancreatic digestion and silver impregnation, saw a concentric arrangement of fibers around the secondary nodule. But within the nodule he encountered only extremely delicate, loosely arranged fibers, which were difficult to trace for any distance. He demonstrated dispersion of the fibers at one pole of the nodule, which he designated as the antrum. This he interpreted as an opening through which the lymphocytes escaped from the nodule.

The observations of Hasskó and myself with neoarsphenamine and silver impregnation gave the following picture of the disposition of these fibers: The clear center of the nodule was almost completely free of fibers. The reticular meshwork surrounded the clear center and no dark zone was to be distinguished with elective stains for fibers only. This means that these zones are merely

6. Foot, N. C.: The Endothelial Phagocyte, Anat. Rec. **30**:15-51 (April) 1925.

7. Hoepke, H.: Ueber die Funktion gesunder und kranker Gaumenmandeln, Ztschr. f. Laryng., Rhin., Otol. **22**:1-16 (Dec.) 1931.

8. Goodale, J. R.: The Endothelial Phagocyte of Tonsillar Ring, J. M. Research **2**:394-404, 1902.

9. Novak, J. F., Jr.: The Structure of the Secondary Nodule of the Tonsil, Arch. Otolaryng. **21**:53-58 (Jan.) 1935.

cellular condensations of a transitional character. We were unable to demonstrate any opening or other sign of rupture in the fibrillary wall around the clear center.

Foerster [10] claimed that the basal membrane at the border between the lymphatic tissue and the epithelium is a structure originating from the reticulum through condensation of the fibers. He expressed the belief that it is a net which is usually closed and folded and that it opens up when lymphocytes pass through. Our observations demonstrate that there are several structures which may be termed a "basal membrane"; for example, with the hematoxylin and eosin stain a delicate line can be seen at the margin of the epithelium. On the other hand, even with a sharp boundary against the epithelium, the reticular meshwork does not show any condensation in this region.

With regard to the reticular projections into the epithelium and their traversing a basal membrane, Glücksmann [11] may be cited. He saw disruption of the basal membrane in the growth of human lung rudiments explanted in vitro and in the growth of "projections" in tumor formation. He cited Heidenhain as having first described in normal ontogenic processes a reduction of the membrane around growing adenomeres.

The basal membrane of the epithelium must be interrupted to let pass the projections described in this paper. It is probably reconstructed rapidly, when the particular portion of epithelium assumes once more a continuous character, after being temporarily reticulized.

In connection with the penetration of the reticulum into the epithelium, I may mention the vascular projections described by Renn.[12] He traced the finest capillaries, beginning with their branching from the subepithelial vessels. In his tangential sections the epithelium offered a cribriform aspect, the holes corresponding to the papillary projections of the submucosa. In the center he saw a capillary vessel surrounded by connective tissue. He emphasized that the depressions of the submucosa between the papillary projections are often mistaken for epithelial papillae. As Renn did not examine the basal reticulum, he could not recognize the true nature of these projections. They contain the small vessels of the reticular framework, as does every other part of the lymphatic parenchyma. Even so, he was correct in objecting to the common concept of an epithelium with regular papillae.

With regard to the grouping of lymphocytes within the epithelium, which is reticulized by these penetrating elements, two previously accepted concepts may be cited. Federici [13] expressed the opinion that between the cells of the stratum Malpighii by compression or chemical action of the migrating cells, round spaces are left, which increase with the arrival of new cells. Mollier [14] saw vacuoles in the protoplasm of the superficial epithelial cells, which, together with intracellular lymphatic spaces, expand, according to his concept, with increasing lymph pressure. Both authors assumed that the new elements actively pass through the intercellular spaces and reach the small cavities in the deeper (Federici [13]) or the

10. Foerster, A.: Die Entwicklung der Gaumenmandel im ersten Lebensjahr, Virchows Arch. f. path. Anat. **241**:418-427, 1923.

11. Glücksmann, A.: Preliminary Observations of the Quantitative Examination of Human Biopsy Material Taken from Irradiated Carcinomata, Brit. J. Radiol. **14**:187-198 (June) 1941.

12. Renn, P.: Zur Funktionsfrage der Gaumenmandel, Beitr. z. path. Anat. u. z. allg. Path. **53**:1-37, 1912.

13. Federici, F.: Sur le méchanism et la significance probable de l'émigration des leucocytes à travers l'epithelium des amygdales palatines, Arch. internat. de laryng. **19**:463-468, 1905.

14. Mollier, S. A.: Die lymphoepithelialen Organe, Sitzungsb. d. Gesellsch. f. Morphol. u. Physiol. **29**:14-37 (July) 1912.

superficial (Mollier [14]) layers of the epithelium. It is clear that the cystlike formation seen in figure 3 *A* does not correspond to any of these spaces, as it extends through the entire thickness of the epithelium, while its connection with the underlying lymphatic tissue is demonstrated in all the impregnated sections (figs. 2 *C* and *D* and 3 *B*).

With regard to the movements of the lymphocytes outside the tonsil, it is interesting, as pointed out by Negus,[15] that in the frog ciliary action conveys mucus and small solid objects from the tonsillar region into the esophagus without the aid of a swallowing action. In man the ciliated mucosa terminates in the lower part of the nasopharynx, and the movements of the cover of mucus are directed only by contractions of the pharyngeal constrictor muscles.

CONCLUSIONS

With the help of previous studies and of the observations reported in this paper, the pathway of the tonsillar lymphocyte may be constructed in the following manner:

Intratonsillar pressure is of basic importance in production of the characteristic grouping of the mobile elements into clear, dark and diffuse zones. The proof was given by tissue cultures (Kelemen and Hasskó [3]). In these experiments a piece of tonsillar tissue survived isolated from its former connection with the organ. With the cessation of the pressure from the tonsil, the clear and dark zones soon disappeared. At this new stage of dedifferentiation the lymphocytes were distributed over the whole piece of tissue in a uniform, equally dense, sheet.

The temporary changes in pressure resulting in production of the likewise temporary structures, the secondary nodules, are effected by rapid alterations in the blood supply. The distribution and structure of the vessels in the capsule, as described elsewhere (Kelemen [16]), are adapted to produce these changes in the turgescence of the entire tonsil. The secondary nodules represent pressure centers from which the mobile elements are forced to leave in the direction of the periphery. The greater number move in the direction of the lowest resistance, against the free surface, the epithelium.

The dual nature of the reticular framework is demonstrated in the following way: One system is not influenced by these temporary changes and can be seen in tissue cultures after the lymphocytes have left the explanted tissue. The result is here a continuous network, where the areas of the secondary nodules cannot be distinguished. The other network, shown by impregnation, remains in close connection with the lymph cells; it becomes conspicuous only with greater concentration of the lymphocytes, outside the secondary nodules. The center of the secondary nodule becomes "clear" after expulsion of the mobile elements.

The intimate association of the cellular and the fibrillary elements of the lymphatic tissue can be utilized in tracing the further pathway. It is shown that in histologic presentation this pathway can be demonstrated more clearly by observation of the fibrillary elements.

Portions of the reticulum, carrying within themselves the mobile elements, penetrate the epithelium, disrupting its basal membrane. At first, in the deeper portions of the epithelium these projections are slender and tubelike, but expand to cystlike formations when they encounter the resistance of the condensed cell

15. Negus, V. E.: The Action of Cilia and the Effect of Drugs on Their Activity, J. Laryng. & Otol. **49**:571-585 (Sept.) 1934.

16. Kelemen, G.: The Palatine Tonsil in the Sixth Decade, Ann. Otol., Rhin. & Laryng. **52**:419-443 (June) 1943.

layer at the surface. By their expansion the epithelium is finally ruptured, and the mobile cells are evacuated into the cryptal lumen. With decreasing pressure and retraction of the conical projections of the fibrillary meshwork, the lymphocytes which did not by this time reach the lumen of the crypt are left behind in the retreating tide. They may be seen undergoing destruction anywhere—isolated, outside the fibrillary cage, between the epithelial layers. The projections, described here, as I think, for the first time, are not to be confounded with portions of the parenchyma between the papillae of the epithelium and can be seen in any "reticulized" portion of the epithelium.

Once the cells have reached the lumen of the crypt, they may be attracted by inclusion bodies. But their migration through the epithelium can be effected without this tropism, as it occurs equally in the embryo and in the late stages of tonsillar involution, in which the crypts are empty. The lymphocytes glide along the threads of fibrin filling the crypt and reach the mouth, and from there the surface of the tonsil. The free ends of the fibrinous fibrils show a tuftlike disposition, which may help to carry the cells to the surface. Because of the location of the tonsils in the depth of the pharynx, the salivary corpuscles, inasmuch as they originate from the tonsils, can hardly be imagined as moving freely in the saliva. They are pulled down by the next swallowing action, with the covering blanket of mucus at the surface of the tonsil, into the esophagus.

Though the capacity of the lymphocyte for active migration is uncontested and was observed in our tissue cultures at the height of its intensity, the entire passage of the cell, through the tonsil, the crypt and the pharyngeal cavity can be explained as directed in a passive manner. Changes in the turgescence of the tonsil initiated by rapid modifications in the blood supply offer the necessary pressure to transport the lymphocyte from the germinal center to the mouth of the crypt along preformed highways.

SUMMARY

The movements of the tonsillar lymphocyte represent in their entirety a passive transportation along preformed highways. The mobile elements in the germinal, or action center are driven by increased intranodular pressure into the meshes of the reticulum. Portions of the reticulum penetrate into the layers of the epithelial cells. These projections of the reticulum carry the mobile elements within themselves. Increasing pressure causes these structures to expand and rupture the epithelium, so that the cells are conveyed into the lumen of the crypt. Here they are carried along threads of fibrin to reach the mouth of the crypt and are helped to reach the free surface of the tonsil by a tuftlike disposition of fibrinous fibrils. Changes in pressure initiated in the blood vessels of the capsule are responsible for the formation of the secondary nodule and the transportation of the mobile elements.

414 Beacon Street.

USE OF DIATHERMY IN SUBMUCOUS RESECTION

ARTHUR C. JONES, M.D.
BOISE, IDAHO

It is well known that occasionally there is rather profuse bleeding at the site of the incision for submucous resection, which is troublesome to stop, and that one has to use suction and epinephrine or attempt to catch the small bleeder with a hemostat. This happened in a submucous operation which I performed several months ago. Earlier in the morning I had been using the diathermy knife for the removal of a small basal cell tumor, and it occurred to me that a bloodless incision for the submucous operation might be made with the cutting blade of the diathermy knife.

All of my submucous operations are performed with the area under local anesthesia induced after a preliminary medication of 1½ grains (0.097 Gm.) of pentobarbital sodium and a no. 1 or a no. 2 injection of a mixture of hyoscine, morphine and cactine. I first swab the mucous membrane with a cotton-tipped applicator saturated with a solution of epinephrine hydrochloride (1 : 1,000) and then dipped into a small jar containing powdered cocaine—not cocaine crystals. The entire mucous membrane is gently swabbed with this mixture, and then I inject at several points a 2 per cent solution of cocaine hydrochloride to which epinephrine hydrochloride solution has been added in the proportion of 1 drop to the drachm (4 cc.).

I make the preliminary incision on the cutaneous side of the mucocutaneous border. This procedure was suggested to me by Dr. Benton Colver, of Glendale, Calif., about twelve years ago, when he was at the Kellogg Sanitarium, at Battle Creek, Mich. The reason for going through the cutaneous area is that injury to the mucous membrane many times leaves a scar which causes crusting and bleeding, as is seen in old scar areas on membranes not operated on. By cutting through the skin side one avoids this after-crusting. In making injections into the membrane I use a 30 gage needle instead of the ordinary 26 gage. This 30 gage needle is as small as a fine sewing needle and can be used in many places about the face and eyes with a minimum amount of pain from puncture. This idea of using the small needle came from the plastic surgeon Dr. George W. Pierce, of San Francisco. (A physician will be surprised at how many friends he can make by using this small needle for all facial and ocular injections.)

I use the Cameron diathermy set, with the rheostat at 5. The cutaneous incision is made with the cutting blade. It is made adequately long and is continued down to the cartilage and through the perichondrium. When the perichondrium is cut through by the hot blade, it is caused to curl back slightly, and there is almost no difficulty encountered in separating it from the cartilage. No time is lost in stopping the bleeding or in beginning the loosening of any adherent perichondrium.

I think beginners in surgery have more difficulty in mastering the technic of a complete submucous resection than that of a complete mastoidectomy.

In closing the incision I use two or three silk sutures on a small curved needle. The plastic surgeon sews all of his intranasal incisions. There is much less tendency for the flap to curl out when it is sutured. I am also careful to make sufficiently large counterpunctures to permit adequate drainage, thus avoiding

Read before the Western Section of the American Laryngological, Rhinological and Otological Society, Inc., Los Angeles, Jan. 31, 1942.

troublesome hematomas. Occasionally the counterpunctures are inadvertently taken care of before the end of the operation. The incision heals as nicely as one made with a scalpel.

I have tried leaving out all packing, have sutured through the septum with catgut and have also used all of the various splints, and I have finally returned to the old type of packing which I started to use nearly twenty years ago—that is, a strip of gauze about 18 inches (46 cm.) long and ½ inch (1.25 cm.) wide impregnated with paste of bismuth, N. F., and gently packed into each nostril. Only one strip of gauze is used, so as to prevent the danger that the gauze may slip into the nasopharynx and down into the trachea. I happen to know of 2 patients who died in one year from aspirating gauze into the trachea. This packing is removed the following morning.

SUMMARY

The use of the diathermy blade shortens the time required for submucous resection. It makes the start of the operation much easier. The incision heals as quickly as that made with an ordinary scalpel.

CLINICAL EVALUATION OF TENDERNESS OF THE MASTOID

HARRY ROSENWASSER, M.D.
NEW YORK

According to Rae,[1] tenderness is one of the most important and most constant signs of disease of the mastoid. There are instances, however, in which tenderness is absent, despite extensive disease in the mastoid bone. There are many factors which may influence or modify this tenderness, and they may be divided into (1) general and (2) local factors.

GENERAL FACTORS

A. *Is the Patient Hyposensitive or Hypersensitive?*—According to Libman,[2] patients are not all equally sensitive to pain and the same disease may cause varying clinical pictures in different persons. On the basis of reaction to pain, Libman divided patients into two groups: (1) hyposensitive and (2) hypersensitive. It is fundamentally important that otologists train themselves to differentiate between these clinical groups. This differentiation can be made fairly accurately by a study of the past history, by speaking at length with the patient and by the use of such methods as pressure over the styloid process of the uninvolved temporal bone to determine the patient's general reaction to pain. For example, when one encounters a phlegmatic person who minimizes all previous illnesses as well as his present illness and in whom firm pressure over the styloid process fails to elicit pain, it is fair to assume that the person is hyposensitive. For this type of patient, therefore, the signs and symptoms must be evaluated accordingly. When such a patient is operated on, there often is found far greater destruction of the mastoid bone than the symptoms and signs might have led one to suspect.

On the other hand, there is the patient from whom minimal pressure over the styloid process elicits a response strikingly out of proportion to the degree of pressure used. In this type, pressure exerted on the normal scalp or over the uninvolved mastoid process seemingly causes great pain. This is characteristic of the hypersensitive subject. Lillie[3] stated: "One should be able to classify the type of patient being dealt with, as to his temperament and his susceptibility to pain." He asserted that an experienced observer can see something in the appearance of a patient which may suggest malingering or which, on the other hand, may indicate that the patient tends to make too little of his illness.

Tenderness is best elicited by using the same degree of pressure on both mastoids. The pressure should be slowly applied with the pad of the thumb, with avoidance of the use of the finger nail, and the facial expression of the patient watched. Not infrequently tenderness will be manifested by wincing, if the attention of the patient tested is distracted elsewhere. Tenderness if real should be

---

From the Otologic Service of Dr. J. L. Maybaum, Mount Sinai Hospital.

1. Rae, J. B.: Some Remarks on the Indications for the Simple Mastoid Operation in the Adult, Tr. Am. Laryng., Rhin. & Otol. Soc. **00**:305-310, 1922.

2. Libman, E.: Observations on Individual Sensitiveness to Pain with Special Reference to Abdominal Disorders, J. A. M. A. **102**:335-341 (Feb. 3) 1934.

3. Lillie, H. I.: Significance of Certain Signs and Symptoms in Cases of Surgical Mastoiditis, Minnesota Med. **10**:280-283 (May) 1927.

persistent. With children it is especially important that efforts be made to divert their attention during the test. This occasionally requires considerable patience and ingenuity.

Young children occasionally react to pressure over the mastoid in a defensive manner, as if at some time during the course of an illness, pressure had been applied in a manner which was painful. As a result, a "pain pattern" frequently persists. When such a patient is reexamined, pressure over the suspected mastoid bone may bring out this defensive reaction and give the erroneous impression that the mastoid is exquisitely tender. To paraphrase this statement, pain frequently is anticipated and the prospect causes flinching to occur before significant pressure is made. The confidence and cooperation of the patient must be gained; otherwise, it is safer to minimize the significance of this sign and attempt to establish the diagnosis from the other available data.

B. *In Cases of Suspected Mastoiditis, How Shall the Responses of a Psychoneurotic Subject be Evaluated?*—In a small group of patients there may be noted a marked disproportion between the degree of complaint and the physical signs of disease. This should not, in itself, be considered sufficient to warrant a diagnosis of a neurosis, since persons vary widely in their sensitivity to pain and in their modes of expression, as well as in their past experiences and training. The diagnosis of a neurosis should be made only on the evidence that the patient is sick emotionally and not because organic disease cannot be demonstrated.

A neurotic subject can simulate tenderness of the mastoid, and in such a patient with possible mastoiditis this may be the deciding factor which may lead to a needless operation. Here is a grave situation which challenges all the diagnostic resources of the most experienced clinician. The neurotic patient with possible mastoiditis at times unwittingly seems to act as if he wished to deceive the surgeon. This is the result of deep-seated anxieties, or of identification with relatives or friends or of unconscious wishes in the direction of self injury. This abnormal behavior should not be confused with malingering. Punitive attitudes, including the stand that the patient is responsible for fooling himself, are not warranted. The otologist must not cooperate with patients who may have an "addiction to surgery" (Greenacre [4]). Temporary general improvement following operation in the absence of any pathologic findings is by no means rare. This is due to the secondary gains from the illness in the feeling of having paid the penalty for guilt and in the acquisition of sympathy.

It cannot be overemphasized that a neurosis can exist side by side with serious organic disease and that at times serious organic disease may cause a latent neurosis to come to the surface. When further observation definitely endangers the life of a patient with suspected mastoiditis, conservatism demands that the performance of a possibly needless operation on the mastoid be risked. If the history and the clinical signs and symptoms give reason to suspect neurotic simulation of tenderness over the mastoid, in the absence of other urgent indications for intervention, one should not hesitate to resort to the aid of a trained psychiatrist. However, since the final responsibility rests with the otologist, he should consider the full report of the psychiatrist as simply one factor to be given its due weight in the final decision.

C. *Does Chemotherapy Influence Tenderness of the Mastoid?*—Since the employment in otology of sulfanilamide, sulfapyridine, sulfathiazole and sulfadiazine, remarkable therapeutic results have been obtained. During the administration of

4. Greenacre, P.: Surgical Addiction: Case Illustration, Psychosom. Med. **1**:325-328 (April) 1939.

these drugs, certain changes in the classic signs and symptoms have been repeatedly observed. One of the most striking is the effect of sulfanilamide and its derivatives on tenderness of the mastoid. Horan and French [5] commented that when mastoiditis does occur in patients treated with sulfanilamide the classic picture is rarely observed and that operation is usually resorted to late in the course of the disease. The following case is illustrative of this.

T. A., a 7 year old boy ill with scarlet fever, complained of pain in the left ear on the third day of illness; a spontaneous purulent discharge developed. Administration of sulfanilamide in adequate dosage was promptly begun. The temperature subsided and the rash gradually faded, but the discharge from the middle ear persisted, though diminished in amount. The mastoid was not tender at any time. The use of sulfanilamide was discontinued after four weeks. On the next day the temperature rose to 102 F. and for the first time the mastoid bone was found to be definitely tender; the discharge became profuse. At operation the boy was found to have an extensively diseased mastoid.

Numerous similar instances have been reported by Maybaum, Snyder and Coleman,[6] Hubert,[7] Rosen,[8] Davis [9] and others, in which chemotherapy admittedly altered the clinical course, masking many of the signs and symptoms of mastoid disease, notably tenderness. It is important, according to Galloway,[10] to know whether the patient has had or is still receiving chemotherapy, for one must now learn clinically how the usual symptoms of mastoiditis can be modified by chemotherapy. It naturally follows that modified criteria of diagnosis must be established, in order that administration of the drugs can be continued for their best effect when complications are imminent and not discontinued, as seems too commonly done, for fear of masking symptoms.

LOCAL FACTORS

A. *What is the Effect of Routine Roentgenography of a Mastoid on Tenderness of the Bone?*—Many years ago a patient was admitted to the Mount Sinai Hospital with the classic signs and symptoms of an acute mastoiditis and roentgenograms of the mastoids were made. The following day, when the case was presented on rounds, the "definite mastoid tenderness" present on admission was absent. It was then pointed out by Dr. Friesner that if the tenderness was genuine (due to bone disease) it would reappear within twenty-four to forty-eight hours. This effect of irradiation on tenderness over the mastoid was described by Goosmann [11] (1919), Ross [12] (1932), Schillinger [13] (1932), Dysart [14] (1939) and others.

---

5. Horan, V. G., and French, S. G.: Prevention of Mastoiditis: A Survey of Six Hundred and Twenty-One Cases of Acute Otitis Media Treated with Sulfanilamide, Lancet **1**:680-682 (April 13) 1940.

6. Maybaum, J. L.; Snyder, E. R., and Coleman, L. L.: Experiences with Sulfanilamide Therapy, Arch. Otolaryng. **30**:557-575 (Oct.) 1939.

7. Hubert, C.: Rôle des traitments sulfamidés dans la thérapeutique des suppurationes auriculaires et de leurs complications, Rev. de laryng. **60**:405-410 (May) 1939.

8. Rosen, S.: Acute Mastoiditis Masked by Sulfanilamide, Laryngoscope **50**:198-199 (Feb.) 1940.

9. Davis, D.: Masking Effects of Sulfanilamide on Mastoiditis, M. Ann. District of Columbia **8**:365-367 (Dec.) 1939.

10. Galloway, J. C.: Chemotherapy, Arch. Otolaryng. **30**:901-915 (Dec.) 1939.

11. Goosmann, C.: Treatment of Post-Operative Mastoiditis with X-Ray, J. Radiol., Omaha **3**:273, 1922.

12. Ross, W. L.: Treatment of Mastoiditis with X-Ray, Radiology **18**:1124-1130 (June) 1932.

13. Schillinger, R.: The Apparent Therapeutic Effect of the Roentgen Ray upon the Course of Acute Mastoiditis, Radiology **18**:763-776 (April) 1932.

14. Dysart, B. R.: X-Ray Treatment of Infections: Review of Literature and Report of Cases of Mastoiditis and Sinusitis, Ann. Otol., Rhin. & Laryng. **48**:433-442 (June) 1939.

Why does tenderness occasionally disappear or become greatly diminished after irradiation of the mastoid? According to Desjardins,[15] lymphocytes, leukocytes and eosinophils constitute part of the cellular response to the irradiation of tissue. He stated the belief that irradiation, by destroying the infiltrating lymphocytes and leukocytes, liberates the antibodies contained in them to be more easily available for defensive purposes than if the cells were intact. Many roentgenologists have felt that the irradiation which results during the making of one or two roentgenograms of a mastoid is totally inadequate as a therapeutic agent. Desjardins, Watkins,[16] Goosmann, Dysart and many others who have had considerable experience with roentgen therapy of inflammatory processes throughout the body have written enthusiastically in its praise and have reported cures of mastoiditis with its use. The disappearance of the tenderness after irradiation can be likened to a temporary masking effect similar to that caused by sulfanilamide, and consequently this effect must be considered in evaluating mastoid tenderness.

B. *How Does the Presence of Inflamed Lymph Nodes Over the Mastoid Bone Influence Tenderness of the Mastoid?*—Small circumscribed inflamed lymph nodes over the tip or antrum of the mastoid process may frequently be extremely tender. They may be inflamed secondarily to an abrasion or scratch in the scalp, an unclean scalp, eczema or nits. Frequently tender nodes over the mastoid process are a prodrome of German measles. Inflamed postauricular nodes may occur in infectious mononucleosis and also in syphilis; as a rule, in the latter they are not tender. The inflammation from the nodes may affect the adjacent periosteum and give the erroneous impression that tenderness of the bone exists. Inflamed postauricular nodes may be present concomitantly with an acute suppuration of the middle ear and simulate tenderness due to disease of the mastoid bone, or they may coexist with a suppurative mastoiditis.

Small, shotty lymph nodes are best felt by gentle palpation. When present, the question as to whether or not the underlying mastoid bone is tender should be decided by making pressure as far removed as possible from the site of the nodes. Adenitis will generally subside with the use of large wet dressings. Persistent tenderness despite subsidence of the inflammation of the glands and soft parts enhances the likelihood of disease of the mastoid. Not uncommonly with subsidence of the inflamed glands all traces of mastoid tenderness disappear.

C. *Does the Type of Organism which has Caused the Infection of the Middle Ear and Mastoid Influence Tenderness of the Mastoid?*—In the majority of cases of acute surgical mastoiditis due to beta hemolytic streptococci, pressure over the mastoid bone elicits a prompt response of pain. This is not true, however, in the majority of the cases of mastoiditis caused by type III pneumococci or by Friedländer's bacilli (Klebsiella pneumoniae) and to a lesser degree in mastoiditis caused by Staphylococcus aureus haemolyticus in adults. Characteristic of the infections caused by pneumococci of type III in adults are absence of spontaneous pain, occasional temporal headache, fairly marked tinnitus, seriously impaired hearing, little or no fever and, in the majority of cases, absence of tenderness over the mastoid on deep pressure. At operation the extent of the destructive process is usually distinctly out of proportion to the clinical signs and symptoms. Profant[17]

15. Desjardins, A. U.: Action of Roentgen Rays and Radium on Eye and Ear, Am. J. Roentgenol. **26**:639 (Oct.); 787 (Nov.) 1931.

16. Watkins, W. W.: Treatment of Inflammations by X-Ray, Southwestern Med. **18**:336-346 (Oct.) 1934.

17. Profant, H. J.: Mastoiditis Caused by Friedländer's Bacillus, Ann. Otol., Rhin. & Laryng. **47**:379-383 (June) 1938.

pointed out that type III pneumococci and Friedländer's bacilli when involving the mastoid bone behave similarly, in that they are extremely insidious and apparently produce no general symptoms and few signs, whereas when these organisms involve the lungs the general signs and symptoms are severe.

One of the reasons advanced to explain the absence of tenderness in infections due to these organisms is the presence of an extremely thick mastoid cortex. I am convinced that this is not the significant factor, because in mastoiditis caused by organisms other than type III pneumococci, Friedländer's bacilli or Staph. aureus in diabetic patients tenderness of the mastoid is much more frequent despite a thick mastoid cortex. A fairly consistent condition encountered in mastoiditis in adults due to type III pneumococci or Friedländer's bacilli is a thick mastoid cortex with the cells under the cortex relatively intact, but as one uncovers the deeper cells, the degree of softening and breaking down of bone becomes much more marked.

Occasionally diabetic patients with extensive disease of the mastoid evidence no tenderness. This is difficult to explain. The true nature of the lowered resistance to infection associated with diabetes mellitus is poorly understood despite a wealth of clinical and experimental information.

The degree of tenderness is only in part related to the type of bone involved. A suppurating mastoid process with a thin cortex and pneumatic cell structure is more apt to be sensitive to pressure than would a sclerotic mastoid or one with an extremely thick cortex (Kerrison [18]) involved in a similar suppurative process.

D. *When in the Course of the Acute Suppuration of the Middle Ear Is the Occurrence of Tenderness of the Mastoid Significant?*—Obviously, the presence of tenderness over the mastoid in a patient with suppuration of the middle ear of three weeks' duration is of infinitely greater significance than tenderness occurring during the first week. It is well known that tenderness over the mastoid antrum is common during the early course of acute otitis media, particularly when a myringotomy is indicated. In this type of disease, after adequate drainage of the middle ear takes place the localized antral tenderness usually disappears within twenty-four to forty-eight hours. Although occasionally the pain and tenderness over the mastoid antrum may persist somewhat longer, this type of tenderness does not necessarily indicate disease of the mastoid bone and certainly is not to be regarded in the same light as tenderness occurring after a suppuration from the middle ear of three weeks' duration. An exception to this is the acute suppuration of the middle ear and mastoid seen occasionally in patients with nephritis and acute leukemia.

Repeated pressing over the mastoid bone of a patient in whose case one cannot certainly decide whether or not the mastoid is tender can conceivably cause a mild traumatic periostitis which may account for tenderness, independent of the underlying process in the mastoid bone.

E. *Erysipelas, External Otitis and a Furuncle in the External Auditory Canal Are Other Local Conditions Which Occasionally Simulate Mastoid Tenderness.*—Clinically it has been noted that for about twelve to twenty-four hours before the typical erythema of erysipelas of the auricle appears there may be a transient nystagmus, hyperpyrexia and tenderness of the mastoid. With the appearance of the typical lesion of erysipelas the nystagmus disappears, but the fever and the local tenderness may persist. The nystagmus is by no means constantly found, but it may occur; and for this reason attention is called to this interesting observation. It is difficult to attempt to evaluate genuine tenderness of the mastoid

18. Kerrison, P. D.: The Indications for Operating in Acute Mastoiditis, Tr. Am. Laryng., Rhin. & Otol. Soc. **11**:210-222, 1905.

in the presence of erysipelas or of local cellulitis. Conservatism, however, demands that the subsidence of the erysipelas be awaited. If the erysipelas is treated with chemotherapy, the effect of the drug on tenderness of the mastoid must be considered.

External otitis and furunculosis of the canal frequently cause tenderness of the mastoid. A furuncle on the posterior portion or the floor of the external canal may cause considerable pain, tenderness, redness and swelling of the tissues of the adjacent mastoid process. This, in turn, may be mistaken for disease of the mastoid, especially if the furunculosis is associated with acute suppuration of the middle ear of two or more weeks' duration. In these instances in which acute external otitis or furunculosis and acute mastoiditis possibly coexist, it has been my policy to treat the acute external otitis or furunculosis and eliminate it as a factor which might be influencing tenderness over the mastoid. Despite the utmost care not to move the auricle one may note tenderness over the mastoid bone. If, as is occasionally the case, it is imperative that a decision be made, the test for tenderness of the mastoid should be made far back from the auricle over the region of the emissary vein. Tenderness limited sharply to the region of the emissary vein is frequently indicative of mastoiditis with perisinal abscess formation.

CONCLUSIONS

Tenderness over the mastoid when present and persistent is one of the most significant symptoms of disease of the mastoid. Its absence, however, does not rule out involvement of the bone. The general factors which must always be considered in deciding whether genuine tenderness of the mastoid is present or not are: (*a*) whether the patient is hyposensitive or hypersensitive; (*b*) whether the patient is neurotic, and (*c*) whether there is a potential masking effect produced by the use of chemotherapy.

Obscuration or simulation of tenderness of the mastoid may be caused by: (*a*) the local effect of irradiation during routine roentgenography of the mastoid; (*b*) the presence of inflamed lymph nodes over the mastoid process, and (*c*) the organism responsible for the infection of the mastoid and of the middle ear especially in diabetic patients. The time factor, i. e., when in the course of the suppuration from the middle ear the tenderness over the mastoid occurs, is significant.

Clinical experience indicates only too frequently that one or more of the aforementioned conditions may coexist, and under such circumstances the decision as to whether tenderness of the mastoid is present is made after the complicating role these factors play in simulating, obscuring or modifying tenderness of the mastoid has been carefully weighed.

101 East Seventy-Third Street.

QUANTITATIVE TUBAL FUNCTION

H. B. PERLMAN, M.D.
CHICAGO

The otologist is continuously faced with the problem of evaluating the function of the eustachian tube, not only in civilian practice but also in industrial and military practice. The lack of simple quantitative tests has made this difficult. A great deal of use is made of the Politzer bag, the eustachian catheter and the bougie. These instruments permit only a qualitative evaluation of tubal function. The results obtained in this way are exceedingly difficult to define. Since normal tubal function is not defined quantitatively, one has no good criteria for recognizing pathologic states of the eustachian tube.

In any consideration of tests for tubal function one must keep in mind that inadequacy of tubal function appears under two separate circumstances and that these must be differentiated. First, there are the common daily demands on the tube that arise from the slow and small change in pressure of the air mass over any given point of the earth's surface. This ordinarily amounts to a change of pressure of a few millimeters of mercury during a period of hours or days. Concurrently, common daily demands arise from the differential absorption of the air gases within the closed cavity of the middle ear by the vascular system, with a reduction in the partial pressures of these gases. This absorption also produces small and slow changes of pressure. Most clinical problems relating to tubal function arise from failure of the tube to overcome these small and slow changes of pressure and adequately to ventilate the middle ear.

In contrast to this normal demand, in a small number of persons there are great and sudden demands made on the tube. These persons are engaged in flying, caisson work or deep sea diving. Equalization of differences in pressure of many hundreds of millimeters of mercury in a matter of minutes is required of the tube under these special conditions. While the tube may fail to meet these great and sudden demands, it may be adequate to meet ordinary demands for ventilation of the middle ear. This difference in response is most strikingly illustrated in persons with an infection of the upper respiratory tract. Tubal failure in such persons is common under the abnormal demands of flying, caisson work and diving. The same persons may have tubal function adequate to meet the ordinary small and slow demands of terrestrial existence.

The development and interpretation of any quantitative test of tubal function must be guided by a consideration of the two different circumstances under which the tube is called on to work.

Quantitative studies of tubal function have been limited. The studies were carried out usually with elaborate equipment. This prevented widespread use of the method by the clinician and limited the value of the observation. Practically all the present quantitative knowledge of tubal function is based on the observations

From the Division of Otolaryngology of the University of Chicago.

This work was done in part under a grant from the Douglas Smith Foundation for Medical Research of the University of Chicago.

of Hartmann [1] in 1879. Part of this work was repeated by Armstrong and Heim [2] sixty years later and translated into equivalents for aviation. These observations concerned the resistance of the tube at rest to positive and negative pressure and are important only for an understanding of the reaction of the tube under the special conditions imposed by flying, caisson work and diving. Hartmann also investigated quantitatively the resistance of the tube to Valsalva's maneuver. As an end point he observed the drum for bulging. I [3] found that this bulging produced a low-pitched "lub" sound that could be heard with an auscultating tube. This fact simplified the test in that no assistant was required to record the pressures. When this sound was used as an end point, a minimum normal resistance of the tube to Valsalva's maneuver was established. This resistance was 20 mm. of mercury. This determination was useful in orienting the condition of abnormal patency of the tube in subjects in whom the pressures for Valsalva's test were well below this minimal normal. The upper limit of pressure needed to open the normal tube in Valsalva's maneuver could not be established, since many normal subjects could not inflate their ears in this manner. Hartmann, and before him Lucae,[4] noted an increased resistance to Valsalva's test when the subject flexed his head, and I found an increased resistance also in the prone position and decreased resistance after exercise, section of the fifth nerve and shrinking of the nose.

Since Valsalva's maneuver does not represent the normal physiologic action of the tube but rather tests the tubal resistance to static pressure in the nasopharynx, the results of quantitative determination with it were of limited value—principally useful in orienting the clinical entity of abnormal patency. Quantitative tests of a physiologic act of the tube were indicated. The resistance of the tube during the act of swallowing is a more important test of its ability to ventilate the middle ear. For the past eighty years it has been known that swallowing reduces the resistance of the tube, thus creating optimum conditions for equalization of differences of pressure between the two ends of the tube. Politzer [5] observed that sound heard at the nose could be made to reappear during swallowing after it had died away. He reasoned correctly that this was due to transmission of the sound through the momentarily opened tube. Since that time it has been widely taught that the tube opens on every deglutition, that is, to practically 0 pressure. However, Gyergyay,[6] using this method and auscultating the external ear for transmission of sound through the opened tube, found that the tube opens only infrequently, remaining closed most of the time, even during swallowing. Zöllner [7] recently reinvestigated this problem of tubal resistance during swallowing. He did not use transmission of sound as the end point, but rather bulging of the drum as observed with a microscopic examination of the ear drum (magnification × 10).

1. Hartmann, A.: Experimentelle Studien über die Funktion der Eustachi'schen Röhre, Leipzig, Veit. u. Comp., 1879.

2. Armstrong, H. G., and Heim, J. W.: Effect of Flight on the Middle Ear, J. A. M. A. **109**:417-421 (Aug. 7) 1937.

3. Perlman, H. B.: The Eustachian Tube: Abnormal Patency and Normal Physiologic State, Arch. Otolaryng. **30**:212-238 (Aug.) 1939.

4. Lucae, A.: Zur function der Tuba Eustachii, Arch. f. Ohrenh. **3**:179, 1866.

5. Politzer, A.: Ueber subjective Gehörsempfindungen, Wien. med. Wchnschr. **15**:1710, 1865.

6. Gyergyay, A.: Neue Wege zur Erkennung der Physiologie und Pathologie der Ohrtrompete, Monatschr. f. Ohrenh. **66**:769, 1932.

7. Zöllner, F.: Widerstandsmessungen an der Ohrtrompete zur Prüfung ihrer Wegseinkeit. Ein neues Verfahren und bisherige Ergebnisse an Ohregesunden und -kranken, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **140**:137-154, 1936.

With this end point and an apparatus that delivered oxygen at controlled pressure to the nose he found that 84 per cent of normal subjects required a positive pressure of 0.5 to 4 mm. of mercury to open the tube during the act of swallowing. These pressures are higher than would be obtained if transmission of sound were used as an end point, since the normal drum has a certain stiffness that must be overcome before noticeable bulging takes place. Recently Van Dishoeck[8] approached the problem of measuring tubal obstruction by studying the secondary effects of inadequate ventilation of the middle ear. He reasoned that if the tube were inadequately ventilating the middle ear a negative pressure would exist there which would impair function of the conducting mechanism (particularly at the drum). If he could vary the pressure on the outer surface of the drum at the same time that he delivered a sound of constant intensity to the ear, he could ascertain the pressure needed for optimum function as reflected in optimum loudness of the delivered sound. The pressure needed for optimum loudness was an index of the pressure prevailing in the middle ear. With inadequate ventilation it would be necessary to have negative pressure on the outer surface of the drum for maximum loudness. He constructed an apparatus for this purpose which he called a "pneumophone" and found his reasoning to be correct. The amount of negative pressure (which he measured in centimeters of water) needed to produce maximum loudness indicated the degree of tubal failure to ventilate the middle ear. Normally ventilated ears were exceedingly sensitive to changes of pressure on the surface of the drum; a change of pressure, either positive or negative, of 1 or 2 cm. of water was immediately sensed by a decrease in the loudness and a change in the quality of the delivered sound. Persons with inadequately functioning tubes were found by this test to have a definite constant negative pressure in the middle ear as great as — 50 cm. of water. Another criterion of inadequate tubal function has been described by Crowe[9] and consists of the secondary effect on the audiogram in tubal obstruction. Loss of hearing for high tones in the audiogram is described as an early observation in tubal stenosis, as in children with obstruction of the tube due to lymphoid tissue. The mechanism of this loss is not clarified by the author, nor does he show any quantitative correlation between the degree of loss of hearing and the degree of tubal obstruction. The loss for high frequencies may find its explanation in the secondary effects on the mass of the conducting mechanism. Edema of the mucous membrane over the ossicles or fluid in the middle ear may have a mass-increasing effect on the conducting mechanism in tubal obstruction. When a vibrating system is changed mainly by increasing its mass and not its stiffness or tension, a differential reduction of response to the high frequencies is to be expected from known laws of acoustic physics. This can be verified on the human ear experimentally.[9a] This criterion of tubal failure (high tone loss) when used by itself is not adequate to define quantitatively the primary and secondary physiologic effects of tubal stenosis. On the contrary, early effects of tubal failure with negative pressure in the middle ear result in increased stiffness of the conducting mechanism, especially reflected in the function of the drum membrane by low tone deafness. A negative pressure of about 15 mm. of mercury is needed to produce a drop of 10 decibels

8. Van Dishoeck, H. A. E.: Das Pneumophon (Ein Apparat zur Druckbestimmung in Mittelohr), Arch. f. Ohren-, Nasen- u. Kehlkopfh. **144**:53-58, 1937.

9. Crowe, S. J.: The Recognition, Treatment and Prevention of Hearing Impairment in Children, Laryngoscope **50**:658-662 (July) 1940.

9a. Perlman, H. B.: Lesions of the Conduction Apparatus, Arch. Otolaryng. **37**:680-690 (May) 1943.

in this region, according to Van Dishoeck. Smaller degrees of negative pressure are therefore not easily recognized in the audiogram. Larger degrees result in formation of serum and changes in the mucous membrane to produce a more striking and constant picture of loss of hearing for high tones. Controlled experimental studies of the effect of a pure increase in stiffness of the drum and conducting mechanism as produced by measured amounts of positive and negative pressure in the external canal show a preferential loss in response to low frequencies. This finding is also in accord with known laws of acoustic physics.

It has been possible to check most of these observations of Hartmann, Zöllner and Van Dishoeck by using suitable apparatus, including a pressure chamber.

OBSERVATIONS WITH THE PNEUMOPHONE IN A PRESSURE TANK

Normal subjects were exposed to known, controlled atmospheric changes of pressure in a pressure tank. A normal subject was first tested with the pneumophone just before the pressure was changed in the chamber. This test gave the usual results of maximum loudness at 0 pressure. The pressure of the air in the chamber was now reduced 10 mm. of mercury (from 760 to 750 mm. of mercury), while the subject was cautioned not to make any palatal movements such as one makes in speaking or swallowing that would permit equalization of pressure between the outer and the middle ear. Pneumophone readings were again obtained, and these readings showed that maximum loudness was sensed at about + 10 mm. of mercury. The pressure of the air in the middle ear remained at 760 mm. of mercury, and that of the air against the outer surface of the drum was artificially kept at 750 mm. of mercury. Therefore, to obtain maximum function an additional 10 mm. of mercury pressure was needed against the outer surface of the drum. This additional pressure was produced by the hand bulb of the pneumophone. A decrease of pressure of 20 mm. of mercury in the chamber gave pneumophone readings of about + 20 mm. of mercury for maximum loudness. Any further drop of the pressure in the chamber caused involuntary opening of the eustachian tube (Hartmann) and limited the amount of positive overpressure that could be produced in the middle ear. With the pressure in the chamber reduced by 10 mm. of mercury the subject was next instructed to swallow and to equalize the pressure in the middle ear with the pressure in the chamber. Pneumophone readings now returned to 0. Next, the pressure in the chamber was brought back rapidly to normal atmospheric pressure, while the subject was again warned not to swallow or otherwise open his tube. Now the air pressure trapped in the middle ear was 10 mm. below the external pressure. This would be comparable to the clinical condition produced by inadequate tubal function. Pneumophone readings now were negative, about — 10 mm. of mercury. That is, maximum loudness was secured when the pressure in the external canal was reduced by 10 mm. of mercury to equal the pressure of the air within the middle ear. Then the pressure in the chamber was again reduced by 20 mm. of mercury. The subject was again instructed to swallow and to equalize the pressure of the air in the middle ear with this reduced external pressure. When this was done 0 readings were obtained. Next, the pressure in the chamber was suddenly returned to normal while the subject's eustachian tube was at rest. Now the external pressure was 760 mm. of mercury and the pressure in the middle ear was 740 mm. of mercury. Pneumophone readings now were about — 20 mm. of mercury. A drop of 30 mm. of mercury after equalization of the pressure in the middle ear produced the condition in which the middle ear pressure was 730 mm. of mercury while the external pressure

was 760 mm. of mercury. This differential on the two surfaces of the drum was distinctly felt as a sense of stuffiness or pressure in the ear. Pneumophone readings now revealed maximum loudness at about — 30 mm. of mercury. These controlled studies gave consistent results on a number of normal subjects and demonstrated that differences of pressure on the two sides of the drum could be detected and quantitatively evaluated with the pneumophone. When, because of inadequate tubal function, negative pressure existed in the middle ear the pneumophone could be used to detect this and measure the amount of negative pressure that existed in the middle ear and against the inner surface of the drum.

With these basic studies as a background a simple unit was devised to define quantitatively the normal and the abnormal function of the eustachian tube. This unit consists primarily of a combination of Zöllner's and Van Dishoeck's apparatus. The source of sound not only is used at the ear but can be introduced into the nose to test for transmission though the tube.

The unit consists of an air-tight chamber from which lead several openings (fig. 1). The subject is connected to one opening through suitable tubing, and

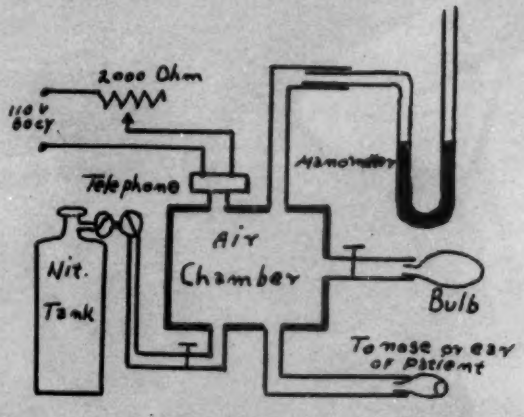


Fig. 1.—Diagram of apparatus for quantitative determination of tubal function. Cutoff valves are used to disconnect the tank of nitrogen or the hand bulb, depending on the test to be performed. The air chamber in the diagram is drawn disproportionately large to indicate clearly the five outlets. The actual size of this air chamber is about 1 by 2 by 5 cm.

an aural and nasal tip provides the desired contact. A mercury manometer is connected to the chamber to record pressures prevailing in the patient connected to the unit. One opening from the air-tight chamber leads through tubing to a tank of compressed nitrogen.[9b] With a suitable regulating valve a constant flow of gas at any desired level can be delivered to the subject. One opening leads to an air-tight telephone receiver, and this receiver is made to produce a 60 cycle tone by driving it with the 60 cycle alternating current of the electric light circuit. The diaphragm of the receiver is perforated so as to obtain its unaltered function under pressure. The intensity of the 60 cycle tone is controlled by a variable resistance. Another opening leads through tubing to a rubber atomizer bulb. This bulb is used to produce small changes of positive or negative pressure in the unit and thus at the ear of the connected patient.

A great deal of positive quantitative information on tubal function can be obtained easily and quickly when the apparatus is used intelligently.

9b. Compressed air is now used instead of the tank of nitrogen.

The following quantitative observations can be made:

1. *The Pressure of the Air Gases in the Middle Ear.*—With the subject connected to the apparatus through a suitable ear piece held firmly in the external meatus, a 60 cycle tone of moderate loudness is delivered to the ear while the examiner varies the static pressure within the external canal with the rubber atomizer bulb (fig. 2). The sound should not be so loud as to fatigue the ear, because judgment of change of loudness would then be difficult. The created pressures against the external surface of the drum are recorded on the connecting mercury manometer. The pressure prevailing when maximum loudness of the 60 cycle sound is noted by the patient represents the difference in the pressure on the two sides of the drum. In normally ventilated ears the change of positive or

Fig. 2.—Photograph of the apparatus in use for measuring the pressure of the air in the middle ear. The tank of nitrogen is disconnected from the rest of the apparatus by closing the stopcock S_1. A 60 cycle sound is developed by a telephone piece attached to the air chamber within the metal box and controlled by the variable resistance (R). This sound is led through tubing into the ear as illustrated. At the same time the examiner holding the rubber bulb (B) varies the pressure in the system and thus against the external surface of the drum. The created positive or negative pressures are recorded by the mercury manometer. The patient watches the mercury column as it is moved above and below a 0 base line and at the same time listens for changes in loudness of the delivered sound. He notes the level of the mercury column when maximum loudness occurs.

negative pressure of 1 or 2 mm. of mercury on the external surface of the drum is associated with a reduction in the intensity and a change in the character of the sound. This judgment is easily made even by children, and consistent readings can be obtained. The impurity of the 60 cycle sound because of higher harmonics permits a change in the character of the sound as well as in its loudness. As the stiff-

ness of the drum is changed by alteration of the pressures in the canal, the response of the drum is selectively affected. That is, the response for low frequencies is more impaired than it is for high frequencies. The higher frequencies of the overtones become more noticeable with respect to the lower frequency of the fundamentals. This differential effect on the conducting mechanism of positive pressure against the drum has long been known. It was studied quantitatively with tuning forks by Fowler[10] and with the audiometer by Van Dishoeck.[11] Another reason for ease in judgment is the rapidity of the changes of pressure produced by the hand bulb. Small and slow changes of loudness with changes in pressure such as are made when one is driving through the mountains are difficult to sense. They stand out only in contrast, as after the act of swallowing when sudden return to normal loudness occurs. Persons with nerve deafness or with almost any conduction lesion not related to ventilation of the middle ear will also sense maximum loudness at a pressure of 0 or of plus or minus 1 or 2 mm. of mercury. Maximum loudness with positive pressure of 2 to 20 mm. of mercury against the drum is sometimes observed immediately after Valsalva's maneuver or politzerization, when air under positive pressure is trapped in the middle ear. The middle ear can be relieved of this overpressure if the subject swallows without holding the nose and if he swallows and holds the nose (Toynbee's maneuver). The former act depends only on the positive pressure in the middle ear for overcoming the minimal tubal resistance. The latter act exerts a more violent force on the tube by increasing the differential in pressure at the two ends of the tube, because a negative pressure (— 1 to — 10 mm. of mercury) is created at the mouth of the tube in addition to the positive pressure trapped in the middle ear. In severe conduction deafness of otosclerosis little or no effect on the loudness of the delivered sound is sensed with alterations in pressure (— 60 to + 60 mm. of mercury) against the drum. This is another way of performing the Gellé test to detect severe fixation of the stapes. No alterations in loudness are sensed in the presence of a perforated drum. This is to be expected, since no differential in pressure can be built up between the two surfaces of the drum. When maximum loudness is sensed at 0 pressure one may conclude that ventilation of the middle ear by the tube is adequate and that the pressure of the air gases in the middle ear is the same as that in the outer canal.

Maximum loudness at negative pressure is an indication of a reduction in the pressure of the air gases in the middle ear due to inadequate tubal function. With just enough negative pressure to restore normal stiffness of the drum and conducting mechanism, maximum function of the conducting mechanism results in maximum loudness. This amount of negative pressure varies from — 4 to about — 35 mm. of mercury and can be maintained because the walls of the middle ear cavity are rigid and the drum offers a certain degree of stiffness. The reduction of pressure in the middle ear in tubal stenosis is brought about with the absorption of the air gases by the blood streaming through the mucous membrane of the middle ear and the mastoid. This absorption is primarily concerned with the oxygen in the air trapped in the middle ear. The partial pressure of nitrogen, carbon dioxide and water vapor in the air in the middle ear is probably not changed in the early stages of tubal obstruction. Because the pressure of oxygen is less in the circulating blood than in the middle ear, oxygen leaves the air and enters the blood. This absorption probably will go on until the pressure

10. Fowler, E. P.: Drum Tension and Middle Ear Air Pressures: Their Determination, Significance and Effect upon the Hearing, Ann. Otol., Rhin. & Laryng. **29**:688-694, 1920.

11. Van Dishoeck, H. A. E.: Measurement of the Tension of the Tympanic Membrane and of the Resistance of the Eustachian Tube, Arch. Otolaryng. **34**:596-602 (Sept.) 1941.

of oxygen in the closed middle ear is halfway between the pressure of oxygen in the artery and that in the vein, or about the pressure of the oxygen in the capillaries. The partial pressure of oxygen in arterial blood is 90 mm. of mercury, in venous blood about 40 mm. of mercury and in the capillaries about 65 mm. of mercury. The pressure in the capillaries is probably about 35 mm. of mercury below the partial pressure of the oxygen in the air of the middle ear normally and represents about the maximum amount of negative pressure that can be developed by the absorption of oxygen from the trapped air in the middle ear by the circulating blood. This figure corresponds with the maximum amount of negative pressure in the middle ear from tubal obstruction as measured with the pneumophone. When the pressure falls below this level serum flows from the blood vessels to help reduce the vacuum in the middle ear and to increase the pressure of the remaining gas by reducing its volume. This can initiate a process whereby all the air in the middle ear and mastoid may go into the blood stream, to be replaced by serum. Various degrees of smaller amounts of negative pressure can be consistently maintained over long periods and reflect a constant degree of tubal stenosis and a resulting constant degree of inadequate ventilation. In such a condition, when the negative pressure becomes greater than the tubal resistance a little air will be sucked up into the middle ear during swallowing. Then the negative pressure will again be equal to the tubal resistance and no further opening of the tube will occur. Most of the lesions studied were acute (weeks) or subacute (months), but tubal obstruction plays a role in chronic otitis media. Quantitative studies of tubal function as described here may clarify this role. Negative pressure readings are sometimes obtained momentarily after Toynbee's maneuver, in which air is sucked out of the middle ear. The next normal deglutition usually restores the pressure to normal.

2. Resistance of the Tube During the Act of Swallowing (fig. 3).—This can be determined quantitatively by several methods with the apparatus. The end point used to mark the opening of the tube is important. Several end points can be used with some differences in the quantitative readings. Transmission of sound at the nose through the opened tube not only is sensed by the subject but can usually be heard by the observer with an auscultating tube. Using this end point, one can observe the opening of the tube with 0 pressure when the subject swallows. At maximum intensity the 60 cycle tone from the receiver is directed into the nose with a suitable glass nasal tip and the ear is auscultated. The opposite nostril is not closed. A constant background of the 60 cycle sound can be heard in the external canal with the auscultating tube and is due to transmitted vibration of the soft tissues and bones of the head. If the mouth of the subject is open, much of the acoustic energy entering the nose will escape through the mouth, but if the mouth is closed the background of 60 cycles is clearly auscultated at the ear. When the tube opens a brief but distinct increase in the "buzz" of the 60 cycle sound entering the middle ear can be heard. This has been recorded experimentally. The transmission of sound can be more clearly auscultated with a short wide tube (12 by ¼ inches [30 by 0.6 cm.]) than with the ordinary tubing (36 by ⅛ inches [90 by 0.3 cm.]). The longer rubber tubing filters out the sound as it travels from the patient to the examiner. The subject can easily detect the momentary increase in loudness of the sound deep in the ear. The only pressure prevailing at the mouth of the tube is the insignificant acoustic pressure of the 60 cycle sound; hence one concludes when these observations are made that the tube opens to 0 pressure during swallowing. With this as an end point it is easy to check the observations of Gyergyay that the normal tube may remain

closed even during swallowing. If the tube does not open during swallowing as tested in this way, then a positive pressure must be delivered into the nose through the same nasal tip along with the 60 cycle sound. The desired positive pressure is obtained by a valve regulating the flow from the tank of nitrogen gas. With the glass bulb in one nostril and the other nostril closed, closure of the nasopharynx by the soft palate during swallowing momentarily blocks the gas from flowing into the pharynx and traps it in the nasal cavity and at the mouth of the tube. When the flow of gas is blocked in this way, its pressure is suddenly raised. This rise in pressure is recorded on the mercury manometer and indicates the amount of pressure prevailing at the mouth of the tube at the moment when the tube resistance is reduced by contraction of the palatotubal muscles. The end point used to mark the flow of gas through the tube may be transmission of the 60 cycle sound through the open tube or bulging of the drum. If the former end point is used lower readings will be obtained than if the latter end point is used,

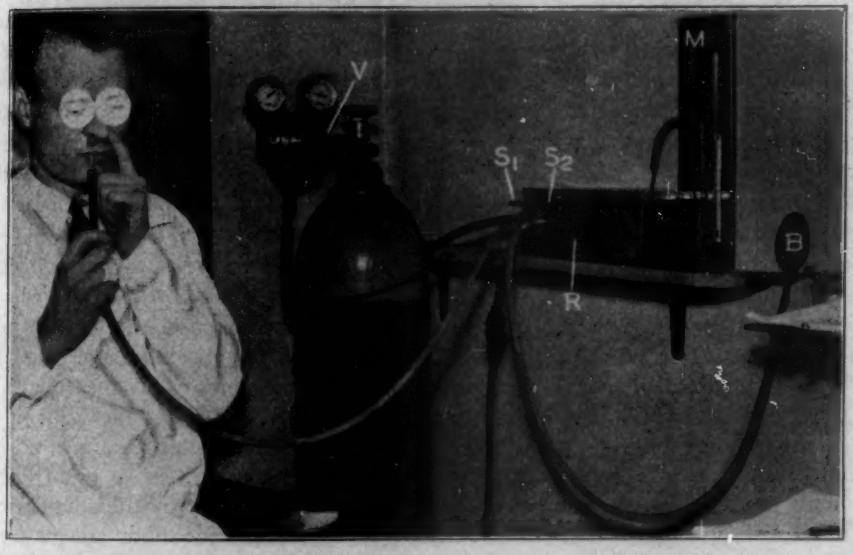


Fig. 3.—Photograph of the apparatus in use for quantitative determination of the resistance of the tube during swallowing. The rubber bulb used for the previous test is disconnected from the apparatus by closing the stopcock S_2. With the needle valve on the regulator any desired flow of nitrogen from the cylinder can be obtained. The amount of pressure that will develop in the nose during swallowing with the opposite nostril closed can be determined beforehand by briefly closing the nasal glass tip with a finger. Blocking the flow of gas in this way raises the pressure within the apparatus, and this increase is registered by the mercury manometer. The 60 cycle sound generated by the receiver is delivered at maximum intensity to the nose at the same time. With an auscultating tube, the brief transmission of the 60 cycle sound through the opened eustachian tube can be heard by the examiner. The pressures prevailing in the nose and therefore at the mouth of the tube are recorded by the manometer. When the drum is observed for evidence of tubal opening, an assistant records the height of the sudden and brief elevation of the mercury column. For quantitative determination with Valsalva's maneuver both the cylinder and the hand bulb are disconnected. The subject simply exhales into the glass nasal tip while the opposite nostril is closed. The pressure that he creates this way at the mouth of the eustachian tube is recorded by the manometer. To detect opening of the tube the ear can be auscultated for the "lub" sound of bulging of the drum or for the transmitted 60 cycle sound, or the drum can be directly visualized for bulging.

because a certain amount of pressure is needed to overcome the stiffness of a normal drum to make it bulge. Bulging is best observed at the annulus about

10 o'clock, with a Siegle speculum. An assistant notes the greatest height of the column of mercury as it is suddenly and briefly elevated. Another way of creating a differential in pressure between the two ends of the closed tube during swallowing (without the use of a source of positive pressure) is to perform Toynbee's maneuver. This creates a negative pressure of a few millimeters at the mouth of the tube and may be sufficient to open the tube. Again, transmission of the 60 cycle sound can be used as a criterion for opening of the tube. Often this amount of negative pressure will be great enough to produce momentary retraction of the drum that can be observed with the Siegle speculum. The negative pressure in the nasopharynx results from bulging of the soft palate into the pharynx as the pressure in the closed oropharynx is suddenly reduced by the increase in the volume of the pharynx. The pharyngeal pressure probably returns to normal with closure of the cricoid sphincter of the esophagus and may be reflected in an immediate return of the drum to its normal contour if the tube has not yet closed. Observations of this nature are alone sufficient to indicate adequate tubal function. The normal tube opens to a positive pressure of a few millimeters of mercury during swallowing. The stenosed tube may require several centimeters of positive pressure to force it to open during swallowing. Clearcut observations can be obtained in unilateral tubal stenosis with exudative catarrh, either on an inflammatory basis or on the basis of obstruction due to adenoid or tumor tissue. The pneumophone readings are corroborated in this way. Another end point that can often be used here is the sudden change of the lines of the bubbles of fluid behind the drum. There is a discrepancy between the recorded amounts of positive pressure needed to open a blocked tube during swallowing and the degree of negative pressure in the middle ear as reflected in the pneumophone readings. This matter needs to be clarified. If one increases the differential in pressure between the two ends of the blocked tube by further exhaustion of the air in the middle ear by Toynbee's maneuver or by delivering a positive pressure of a few millimeters of mercury to the mouth of the tube during swallowing, tubal resistance should be overcome. But it is probably overcome only to a degree that allows a brief molecular flow of gas through the tube. (There are about 27×10^{18} molecules in 1 cc. of gas.) This flow may be adequate to prevent a further drop in pressure in the middle ear, but the tubal opening is not sufficiently wide or lasting to allow adequate air flow for good transmission of sound or bulging of the drum. Experimentally with a microphone in the external canal it is possible to show that even in normal tubes, the period of opening during deglutition as tested by sound transmission may vary from about $\frac{1}{60}$ to $\frac{1}{3}$ of a second. To obtain audible transmission of sound or bulging of the drum in tubal stenosis a sustained and wide opening of the tube is required. This is achieved by delivering a greater amount of positive pressure at the nose. The normal tube does not require this amount of positive pressure to allow transmission of audible sound or movement of the drum. Similarly, there is a discrepancy between the pneumophone readings in uncomplicated infections or allergic rhinitis and the amount of pressure needed to open the tube widely in these conditions during swallowing. Here, again, opening sufficiently to obtain a molecular exchange of air to overcome the small and slow changes of pressure can explain the normal or small negative pressure in the middle ear that is observed with the pneumophone. Considerable positive pressure is needed to open such a blocked tube widely and long enough to permit bulging of the drum, transmission of sound or rapid equalization of large differences of pressure such as occur when one is flying or going through an air lock.

At the same time that the examiner has determined quantitative resistance of the tube during the act of swallowing he has carried out an inflation of the diseased ear in an easy and painless manner. This, of course, results in a sudden improvement of function noted by the patient. The inflation is carried out with just sufficient pressure and does not expose the ear to excessive pressures produced by other methods of inflation. Moreover, repeated inflation during one sitting of the patient can be carried out and insures a greater amount of exchange of gases between the middle ear and the external atmosphere. Inflation carried out with nitrogen produces more sustained improvement of function than inflation with ordinary air, since the gas is more slowly absorbed than is oxygen (half as fast according to Van Dishoeck). The more rapid absorption of oxygen along with replacement of normal air of the middle ear by prolonged inhalation of mixtures

TABLE 1.—*Examples of Tubal Obstruction*

Cause of Obstruction	Name	Age	Sex	Mm. Hg Maximum Loudness, Pneumophone	Mm. Hg Required to Open Tube on Swallowing	Mm. Hg Required for Valsalva's Test	Comments
Infection, acute	B	63	F	—10	50	60 neg.	After inflation pneumophone reading 0
	W	32	M	—14	40	60 neg.	Recurrence of negative readings 30 minutes after inflation
	H	40	M	—20	14	.....	After inflation Toynbee reaction negative; opposite normal ear gave 0 reading on pneumophone
Infection, chronic	W	19	M	—40	60 neg.	.....	Catheter needed to inflate tube; after inflation pneumophone reading +20; suppuration of attic
	D	36	F	— 8	40	50 neg.	
	F	71	F	—20	30	30 neg.	After inflation pneumophone reading 0
	J	45	M	—20	50	40 neg.	Opposite ear normal
Adenoid	D	16	M	— 8	38		
	D	6	F	—24	40	.....	After inflation pneumophone reading +10
	D	28	M	—20	70		
	D	5	F	—20	20	.....	Lymphoid tissue in mouth of tube
	B	7	F	—10	20	20 neg.	After inflation Toynbee reaction negative; pneumophone reading +10
Tumor	J	55	F	....	26, 24 L	.....	After inflation Toynbee reaction negative; pneumophone reading +10
	W	57	F	....	64 R neg.	.....	Right tube more severely blocked by carcinoma than left
	H	56	F	....	6 L 40 R neg.	.....	Carcinoma about right vault; exudative catarrh in right ear

with an abnormally high oxygen content predisposes to formation of serum in the middle ear after flying at high altitudes. In such cases Poppen [12] found that air of reduced oxygen content forced into the middle ear by Valsalva's maneuver after the subject had held his breath produced more lasting improvement than did ordinary air. This is based on the same reasons. After the inadequately ventilated middle ear has been inflated with the apparatus, pneumophone readings are consistently and definitely changed toward normal or 0 readings, or if a definite positive pressure has been created in the middle ear, readings may become positive. If the obstruction of the tube remains unrelieved, the pressure in the middle ear will again become negative as tested with the pneumophone. Usually this occurs in a few hours. In the presence of unilateral conduction deafness the 60 cycle sound introduced into the nasal cavity is continuously heard in the affected ear as a referred Weber response. This should not be confused with the brief direct transmission through an open tube which can be objectively detected by the examiner. The amount of pressure that can be delivered to the tube during swallowing is

12. Poppen, J. R.: The Ear in Flying, Laryngoscope **51**:974-982 (Oct.) 1941.

limited by the strength of the palatal muscles closing the nasopharynx. When the suddenly raised pressure created in the nasopharynx is sufficiently great, the palatal muscles will be blown away from the nasopharynx, and this will allow the pressure to be dissipated into the pharynx. If the flow of gas delivered to the nose before swallowing begins is too strong the palate cannot rise against it at the beginning of the act of swallowing to a degree necessary to close the nasopharynx. The strength of the palatal muscles as tested in this way measures about 100 to 120 mm. of mercury. This amount represents, therefore, the upper limit of pressure that can be delivered to the mouth of the eustachian tube during swallowing. But in practically every case of tubal stenosis opening of the tube during swallowing can be induced with pressure well below this level.

3. *Quantitative Determination of Valsalva's Maneuver.*—Several end points can be used to detect opening of the tube in this procedure. The drum can be observed for bulging most conveniently with a Siegle speculum. The ear can

TABLE 2.—*Examples of Unobstructed Tube*

Diagnosis	Name	Age	Sex	Mm. Hg Maximum Loudness, Pneumophone	Mm. Hg to Open Tube on Swallowing	Mm. Hg for Valsalva's Test	Comments
Normal	T	28	F	0	2	50	Toynbee reaction positive
	R	49	F	0	0	20	60 cycle transmission auscultated
	F	38	F	0	3	24	Toynbee reaction positive
Otosclerosis	H	25	M	0	0	..	60 cycle transmission through tube auscultated
	G	22	F	0	4	..	Toynbee reaction positive
	L	26	F	0	6	..	Toynbee reaction positive
	L	35	F	0	2	..	Toynbee reaction positive
Severe otosclerosis	F	61	F	−20 to + 20 neg.		..	No change in loudness with pressure (reaction to Gellé test positive)
	V	34	M	−20 to + 20 neg.		..	No change in loudness with pressure (reaction to Gellé test positive)
Abnormally patent tube	C	40	M	0	0	0	Open tube closed in prone position; then pressure for Valsalva maneuver 10 mm. powder Valsalva maneuver negative to 40 mm. Hg
	P	40	M	0	1	10	Inspiration with mouth closed emptied middle ear; tube closed in prone position, opened on deep respiration
	K	35	F	0	3	0	

be auscultated for a low-pitched "lub" sound that marks the moment the drum suddenly bulges as the tubal resistance is overcome and air enters the middle ear. The ear can be auscultated for the transmission of the 60 cycle sound. Quantitative tests for Valsalva's maneuver are particularly useful in recognizing abnormal patency of the tube but also corroborate the findings of the two previous tests in tubal stenosis. Practically no person with a blocked tube as measured by the pneumophone or during the act of swallowing could inflate his ear by Valsalva's method. Any reading below 20 mm. of mercury may be considered as indicating an abnormally patent tube. When the tube is continuously open, auscultating the ear for transmission of the 60 cycle tone at the nose easily corroborates the autophony described by the patient. Then closure of the tube by pressure on the neck, by the subject's assumption of the prone position and by insufflation of boric and salicylic acid powder, can be immediately detected with the auscultating tube by the change in the transmitted sound at the ear.

4. *Quantitative Determination of Toynbee's Maneuver.*—The creation of negative pressure at the mouth of the eustachian tube increases the differential in pressure between the inner and the outer end of the tube. This difference in

pressure, may in itself be great enough to overcome tubal resistance during the act of swallowing. In a normal ear an excess of pressure in the middle ear over the outer pressure at the nasopharynx of 15 to 30 mm. of mercury is sufficient to open the tube even when it is at rest and palatal muscles are not in action. During the act of swallowing this tubal resistance is greatly decreased and a smaller amount of excess pressure in the middle ear will open the tube. If the tube opens only long enough to allow air to be sucked out of the middle ear, the drum will be retracted. If the tube opens long enough to transmit the negative pressure and the immediately following normal pressure, then the drum will be seen to move inward and then immediately back to its resting state. Toynbee's maneuver results in the momentary creation of negative pressure in the nasopharynx of 1 to about 10 mm. of mercury as measured with this apparatus.

SUMMARY

Intelligent use of an apparatus combining a controlled source of positive pressure, a source of sound, a hand bulb with which to create small positive and negative pressures against the drum and a mercury manometer permits a rapid and simple evaluation of tubal function in a quantitative way. Changes in tubal function due to physiologic or therapeutic factors can be recognized and quantitatively defined.

Controlled observations in a pressure chamber demonstrated that accurate information about the differences in pressure on the two sides of the drum could be obtained with the apparatus.

These quantitative observations help to identify the normal tube, the abnormally patent tube and the stenosed tube. The degree of stenosis can be defined quantitatively.

The apparatus permits easy inflation of the middle ear when the tube is stenosed and thus avoids instrumentation. One uses just the amount of pressure needed instead of uncontrolled excessive pressure. This inflation can be repeated many times at each sitting to obtain maximum ventilation of the middle ear.

950 East Fifty-Ninth Street.

VOICE PRODUCTION AND THE LARYNGOLOGIST

WILLIAM A. C. ZERFFI
NEW YORK

A few years ago Dr. Chevalier Jackson made the following assertion: "In 98 per cent of cases the professional singer ends up with the loss of his voice." Coming from so eminent a source, this statement should give professional singers something to think about, and it should also have a profound effect on laryngologists, for since laryngology pertains to the study of the larynx and singers sing with the larynx, laryngologists cannot escape a certain degree of responsibility for this truly lamentable state of affairs. No doubt the laryngologist may feel that his responsibility ends when he takes care of pathologic matters—but does it? Are there not many conditions which are created directly by improper use of the voice and which, if neglected, lead to the loss of voice which Dr. Jackson predicted? However, in defense of the laryngologist it must be emphasized that singers have come to believe that the production of the voice, i. e., the functioning of the larynx in tone production, is a field which lies outside the province of the physician, and they would not be likely to listen to any suggestions which did not emanate from a teacher of singing. And it is only too true that the laryngologist seldom feels that he is sufficiently well acquainted with the subject of voice production to be able to offer sound advice.

This situation places the singer in a sort of no man's land, with the vocal teacher on the musical side and the laryngologists on the medical side, neither of the two being particularly anxious to enter into this arid and barren territory.

The great majority of laryngologists are well versed in their specialty up to a certain point, that being the functioning of the larynx in tone production. As much can rarely be said of the teacher of singing. Since there are no definite requirements established for such teachers, whatever preparation they bring to their work is entirely individual. Some have studied with other teachers or other singers, but the principle which teachers of singing seek to inculcate in their pupils is generally based on the assumption that if a singer sings well, particularly if he has been a successful singer, he knows how he sings and can pass this knowledge on to others. That is a principle which if applied to medicine would lead to the assumption that the man with a good digestion automatically becomes a stomach specialist and the man with good eyesight an oculist. The absurdity of this attitude as applied to medical matters is only too obvious, but in singing it has become hallowed. The possession of a good voice is frequently considered sufficient equipment with which to teach singing and even an ability to play the piano is often believed to be all that is necessary in order to train singers for the exercise of their profession.

However, despite all this, one inescapable fact remains, and that is that the singer sings with an organ which is an integral part of his body, and bodily organs certainly belong in the province of the physician. The orthopedic surgeon can hardly be classed as a specialist in the art of walking or of dancing, but his knowledge of anatomy and of physiology qualifies him to pass judgment on a type of movement which is not in accordance with sound physiologic principles and which may entail unfortunate consequences. It is hardly necessary to emphasize that improper use of the larynx also brings a train of unfortunate consequences, but it

seems to be accepted that all this has nothing to do with the laryngologist. While it is true that some investigators have endeavored to solve the problems which confront singers and speakers, in general it must be said that so far little has been offered which has real scientific worth. Two notable exceptions come to mind. One is the valuable analysis of the voice by the late Hudson Makuen,[1] who recognized the existence of the two separate mechanisms involved in the production of the voice, namely, the tone-producing mechanism (larynx) and the articulating mechanism (pharynx, tongue, palate and related structures). Another important contribution was made by Dr. Elmer L. Kenyon,[2] who analyzed vocal problems relating to speech with keen insight and gave a highly effective method for the elimination of vocal troubles. He advocated that the examiner palpate the larynx with the finger in order to determine whether the vocal cords were being allowed freedom of action, a method which has been used by me in voice production for song and for speech since 1912. Dr. Kenyon described this method as "the one objective means, aside from the limited possibility of study of the vocal cords themselves by means of the laryngoscope, by which the correctness of action of the vocal cords may be understood. More exactly it tells whether the action of extrinsic musculature is normal; from this we may infer whether the vocal cords are hampered in action or act freely, whether tension is present and whether the mechanism of pitch production is normal or disturbed in action." This statement was made in a paper read before the Section on Laryngology, Rhinology and Otology at the Seventy-Ninth Annual Session of the Americal Medical Association in Minneapolis, June 15, 1928.

It might be thought that the presentation of a system which would make the diagnosis of improper production of the voice relatively simple would have been hailed by laryngologists, particularly as the diagnosis could be achieved without any complicated involvements such as knowledge of voice production or of phonetics, but unfortunately it does not seem that its importance was realized. When, in 1939, I[3] outlined my own and somewhat similar procedure for the detection of incorrect use of the voice, Dr. Joel J. Pressman,[4] in a review of my article dismissed the procedure, saying, "I tried it and it doesn't seem to work," and thereupon modified this statement by saying that his impressions might perhaps change "after the necessary years of study with the finger in the approved position."

One who has been in the vocal profession for many years has become accustomed to the inaccurate and the meaningless statements so frequently made by vocal teachers. As has already been said, their preparation is usually such as to preclude the development of a scientific attitude. But it was a shock to find Dr. James Sonnett Greene[5] expressing himself in a discussion of what he called "psychophonasthenia," as follows: "Paradoxical as it may seem, these patients do not suffer from a weak larynx or from functional voice fatigue, as is pointed out by Flatau, Froeschels, and others, but from a larynx which in reality is over-powerful. The patient uses his larynx as an instrument of pressure instead of an instrument of performance. He inhibits voice production through muscular pressure, vocalizing *with* his throat instead of *through* his throat." If this last phrase, *"with* his throat instead of *through* his throat," had emanated from a teacher of singing it

1. Makuen, G. H.: Defects of Speech and Voice. Am. Phys. Educ. Rev. **13**:32 (Jan.) 1908.
2. Kenyon, E. L.: Action and Control of the Peripheral Organs of Speech, J. A. M. A. **91**:1341 (Nov. 3) 1928.
3. Zerffi, W. A. C.: Functional Vocal Disabilities, Laryngoscope **49**:1143 (Nov.) 1939.
4. Pressman, J. J.: Physiology of the Larynx, Laryngoscope **50**:277 (April) 1940.
5. Greene, J. S.: Psychiatric Therapy in Dysphemia and Dysphonia: Stuttering, Psychophonasthenia, Aphonia, Falsetto, Ann. Otol., Rhin. & Laryng. **47**:615 (Sept.) 1938.

might be forgiven, but it can hardly be considered worthy of any one with scientific training. No matter which way the matter may be twisted or turned, the sounds made in the larynx are made by the use of the vocal cords. To speak of vocalizing through the throat and not with it simply does not describe what actually takes place when a person phonates. It is inconceivable that a patient afflicted with speech difficulty could have developed an overpowerful larynx. Weakness of voice is almost invariably caused by overuse of the external muscles of the throat, which by taking over some of the work which should have been relegated to the internal or true vocal muscles bring about deterioration of their own strength.

Turning now to an article by Dr. Emil Froeschels,[6] one finds him speaking of "Voice Hyper-Functions" as follows: "I found seven kinds of hyperfunctions located in seven different spots." Starting with the second type, Dr. Froeschels described it as the "coup de glotte." The third he described as a too violent contraction of some pharyngeal constricting muscles, the fourth as a retraction of the posterior part of the back of the tongue and of the root of the tongue, the fifth as a hyperfunction of the soft palate, the sixth as a stiffening of the blade and of the tip of the tongue and the seventh as a stiffening of the lips. As to the first type, that was described as "the air being pushed violently from below against the vocal cords." Dr. Froeschels has accurately described these hyperfunctions. Disregarding the stiffening of the lips, easily observable and easily corrected, I would point out that the six other hyperfunctions can immediately be detected and eliminated in due time by the use of the method of finger palpation advocated by Dr. Kenyon and by me. They can be so satisfactorily eliminated by this method that they need not be considered as separate problems.

Enough has been said to indicate how vitally necessary it is for the solution of the problems which confront the singer and the speaker that laryngologists and teachers of voice production pool their knowledge and openly discuss these problems. Advances in the science of voice production have been so pitifully meager since the days when Garcia gave the world his laryngoscope that keen embarrassment should be felt on both sides. The gravity of the situation can hardly be exaggerated. Singing careers are cut short and often lives are wrecked for lack of rational training, and sufferers from faulty speech habits drag themselves around in what can only be described as acute misery, handicapped in every direction. There is no doubt that some speech difficulties are due to psychologic factors, but I believe those are greatly in the minority. Once the voice functions well physically, confidence in the ability to use it returns, and even the dread nervousness which so often assails the singer as well as the speaker disappears when it is found that the voice will respond to the demands made on it.

31 West Eighty-Fourth Street.

6. Froeschels, E.: J. Speech Disorders **5**:2 (March) 1940.

ANATOMY OF THE TRACHEOBRONCHIAL TREE FROM THE BRONCHOSCOPIC STANDPOINT

STUDY OF TWENTY PAIRS OF LUNGS; PREPARATION OF LUNGS FOR DEMONSTRATION

E. W. HAGENS, M.D.
CHICAGO

An examination of textbooks and of the literature shows that a number of valuable studies have been made on the anatomy of the lung. They include the usual dissections and special casts of the lung, such as those reported so beautifully by de Pablo [1] in 1940. However, when the bronchi are considered more strictly from the bronchoscopic point of view, there is much less to be found. Several years ago Huizinga and Behr [2] reported an investigation of 125 pairs of lungs from this point of view. They described the usual bronchial tree and gave their paper the title "Systematic Bronchoscopy." While studies of the anatomy of the lung from the various points of view are necessary and most valuable to the endoscopist, nevertheless none but those doing such work view the bronchi in the endoscopic manner. In recent years endoscopy has become more and more useful to the internist, the thoracic surgeon and the pediatrician. As a result the endoscopist has of necessity been forced to extend his knowledge of the structures within the chest.

While it is possible to see only to a certain point down any of the bronchi, it is evident that to know the usual bronchial divisions and their general directions may be of great help in localizing pathologic conditions. It was with these thoughts in mind that 20 pairs of lungs from cadavers were dissected and measurements taken. It is evident that measurements of organs from cadavers cannot be considered as accurate as those of living organs; however, they represent an approximation of the true measurements and give an idea as to the relative dimensions of the various bronchi. Unfortunately, the sex and the race of the cadaver from which the pair of lungs was taken could not be obtained in enough instances to be of value.

The following is a résumé of the dissections:

Trachea.

Length 11 to 12 cm.

Diameter 18 to 25 mm. horizontal by 15 to 19 mm. vertical—av. 20 mm. horizontal by 17 mm. vertical. Cartilaginous rings are absent on the dorsal (posterior) surface.

Right Lung.

Right Main Bronchus.

Comes off laterally at about a 25 degree angle from the trachea.

Length—av. 2.5 cm. (one specimen 3.2 cm., one 3.5 cm. and one 4.5 cm.).

Diameter—av. 14.5 mm. horizontal by 12 mm. vertical.

From the Department of Otolaryngology, Northwestern University Medical School.

1. de Pablo, V. E.: La arquitectura bronchopulmonar, Buenos Aires, A. Guidi Buffarini, 1940.

2. Huizinga, E., and Behr, E.: Systematic Bronchoscopy, Acta oto-laryng. **28**:67-73 (Jan.-Feb.) 1940.

I. Right Upper Lobe Bronchus.

Comes off at the level of the carina as a rule; may come off 1 cm. lower.
Diameter at orifice—av. 9 mm.
Divides after 2 to 12 mm. (av. 5.5 mm.) into three branches.
Variation: Four specimens had two branches; one had four.

II. Right Middle Lobe Bronchus.

Diameter at orifice—av. 3 by 7 mm.
Courses downward somewhat ventrally and laterally.
Divides after 12 to 35 mm. (av. 20 mm.) into two branches: One is to the more medial and ventral part of the lobe; the other, to the more dorsal and lateral part. The spur is usually more or less horizontal.
Variation: While the bronchus is always present, the lobe is often incompletely separated, and in five specimens there was no middle lobe.

III. Right Lower Lobe Bronchus.

Av. diameter at orifice 7 by 8 mm.
Divides into:

1. Right dorsal branch.

Comes off the dorsolateral wall immediately.
Diameter at orifice usually about 3.5 mm.
Divides after 2 to 7 mm. into two branches. The spur is usually 12 to 6 o'clock.
Variation: In two specimens an extra dorsal branch was found. The larger came off just proximal to the start of the lower lobe bronchus; the other, just distal to it. In one specimen the dorsal branch divided into three branches.

2. Right medial (cardiac) branch.

Comes off 3 to 15 mm. (av. 8 mm.) from the start of the right lower lobe bronchus.
Comes off on medial wall of right lower lobe bronchus.
Av. diameter at orifice 3 mm.
Courses downward along the median border of the lower lobe.
Divides after 2 to 25 mm. (av. 13 mm.) into two branches, the spur being more often horizontal than vertical.
Variation: In one specimen this bronchus supplied a fourth (azygos) lobe.

3. Right main continuation.

Diameter about 5 to 6 mm. at orifice.
Divides variously:
(*a*) Frequently the first branch comes off the lateral wall 7 mm. from the medial branch (or 15 mm. from start of right lower lobe bronchus). This branch courses laterally and down to supply the more lateral part of the lobe.
(*b*) The second branch may be another lateral branch or a medial one from the median wall.
(*c*) The main bronchus then continues on downward, branching variously, to supply the median lower part of the right lower lobe.

Left Lung.

Left Main Bronchus.

Comes off at about a 45 to 50 degree angle from the trachea.
Length—av. 5 cm.
Diameter—av. 14 mm. horizontal by 10 mm. vertical.

I. Left Upper Lobe Bronchus.

Comes off laterally about 5 cm. from the carina.
Diameter at orifice 8 by 9 mm.
Divides after 2 to 25 mm. (av. 12 mm.) into two branches.
Variation: In one specimen the lower branch of the left upper lobe bronchus supplied a partially separated third lobe.

II. Left Lower Lobe Bronchus.

Diameter at start 6 by 9 mm.

Divides into:

1. Left dorsal branch.

Comes off the dorsal wall 2 to 12 mm. (av. 6 mm.) from the start of the left lower bronchus.

Av. diameter 4 mm.

Divides after 2 to 15 mm. (av. 5 mm.) into two or three branches. If there are two branches, one of these usually divides again.

Variation: In two specimens a smaller additional dorsal branch arose more distally.

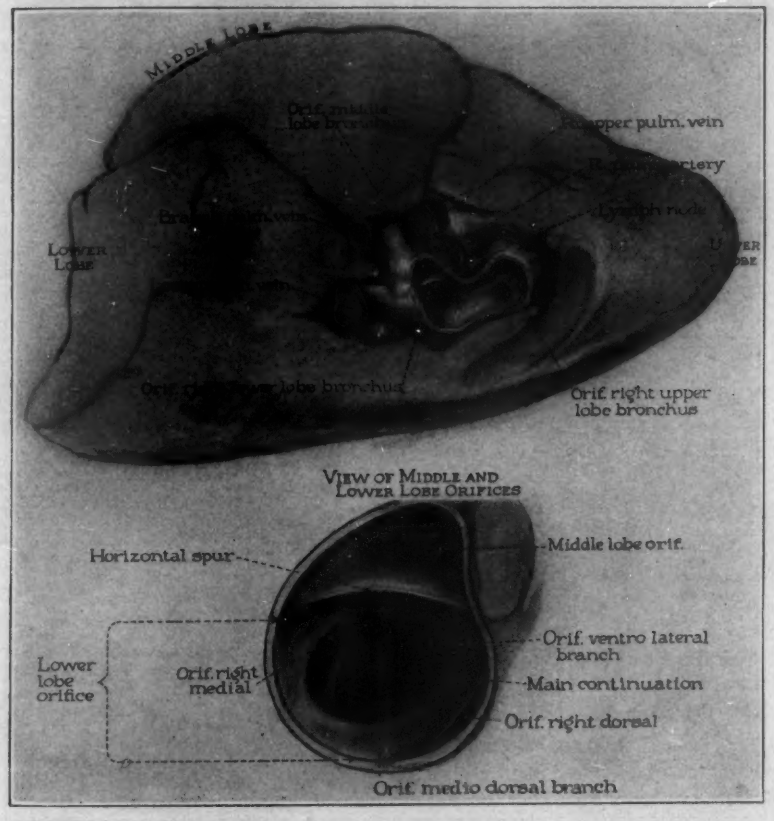


Fig. 1.—Hilar view of the right lung.

2. Left lower lobe continuation.

Courses downward and laterally and, about 14 mm. from the left dorsal branch (20 mm. from start of left lower bronchus), divides by a horizontal spur into two branches (*a* and *b*).

a and *b* course downward, *a* being more ventral and supplying the more lateral part of the lobe, while *b* supplies the more medial part.

a frequently has a lateral branch after about 8 mm.

b may have a lateral branch after a few millimeters and frequently has a horizontal spur after a short distance.

Variations: In two specimens a medial branch was found (corresponding to that on the right side) between the left dorsal branch and the usual division of the left lower lobe continuation.

The drawings (figs. 1 and 2) and the sketch (fig. 3) give visual form to the observations on the bronchial tree. The drawings show the hilar region of each lung, while the sketch helps to visualize the bronchi as the endoscopist encounters them. It also indicates the main branches that lie beyond what can be seen through the bronchoscope.

SOME GENERAL OBSERVATIONS ON RIGHT LUNGS

1. While the right lung usually has three lobes, these may be incompletely separated. In five specimens only two lobes occurred, the usual upper and middle

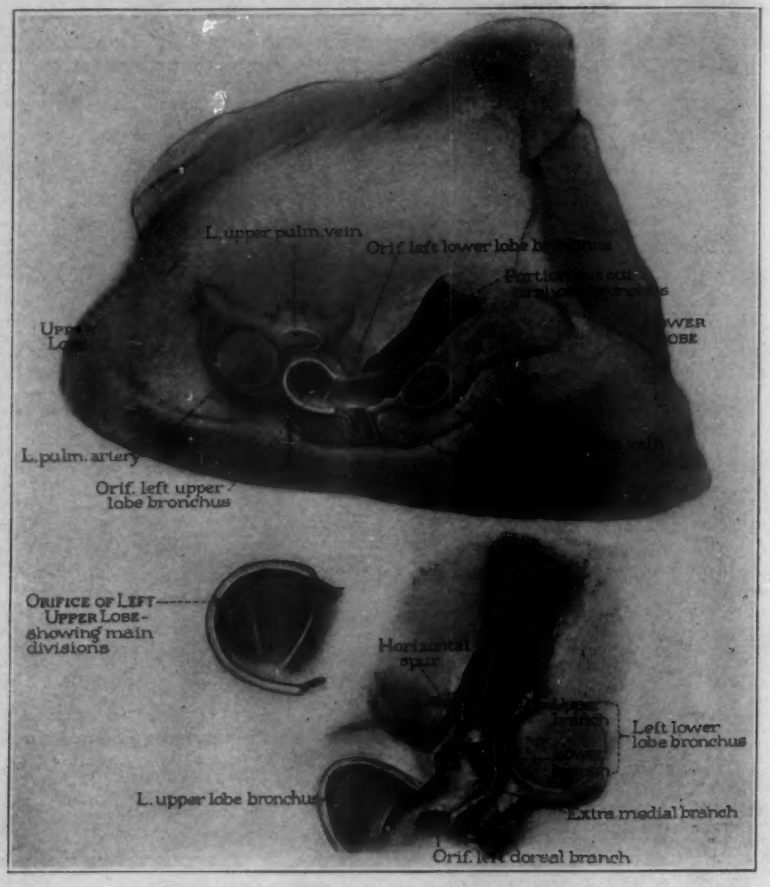

L. upper pulm. vein
L. pulm. artery
Orif. left upper lobe bronchus
Orifice of Left Upper Lobe - showing main divisions
Horizontal spur
Left lower lobe bronchus
L. upper lobe bronchus
Extra medial branch
Orif. left dorsal branch

Fig. 2.—Hilar view of the left lung.

lobes being represented by a single upper lobe. In one specimen four lobes were found, the fourth being a medial or azygos lobe.

2. The right upper lobe bronchus usually divides into three branches. In four specimens there were only two, and in one lung there were four branches.

3. The right middle lobe bronchus is always present even though there is no middle lobe. In the latter case it supplies the lower part of the upper lobe. The bronchus divides into two branches after a varying distance.

4. The right lower lobe bronchus usually branches into (*a*) a dorsal branch, coming off immediately, (*b*) a medial branch, coming off within a centimeter and (*c*) irregularly placed branches from the main continuation downward.

SOME GENERAL OBSERVATIONS ON LEFT LUNGS

1. The left lung usually consists of two lobes.

2. The left upper lobe bronchus divides into two branches.

3. The left lower lobe bronchus generally divides into a number of branches. A dorsal branch (*a*) comes off within a centimeter of the orifice. It supplies the upper part of the left lower lobe. The main continuation after about 2 cm. divides into two rather equal branches (*b* and *c*). These are separated by an approximately horizontal spur, and they continue downward, one above the other, to supply the basal part of the left lower lobe. Figure 3 shows these divisions in a diagrammatic form. Thus it may be concluded that the human lung has a fairly definite pattern of bronchial division. If studied, it provides the endoscopist with considerable information and aid. Variations do occur as to number and size of lobes and as to size and mode of bronchial branchings. The knowledge

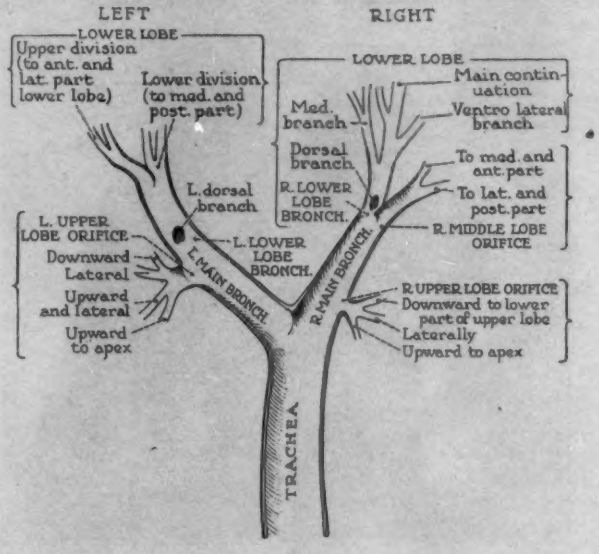


Fig. 3.—Topography of human lungs from the bronchoscopic point of view.

of these variations may be of great importance to the endoscopist, the roentgenologist, the internist and the thoracic surgeon.

SOME GENERAL OBSERVATIONS IN REGARD TO THE BLOOD VESSELS ABOUT THE HILAR REGION

The aorta crosses anterior to the trachea, just above the bifurcation of the latter, and after turning down passes underneath the left main bronchus. The left pulmonary artery, as well as the upper left pulmonary vein just below, cross anteriorly over the left main bronchus. The lower left pulmonary vein courses anteromedially to the left lower lobe bronchus (fig. 2).

On the right side the right pulmonary artery passes anteriorly across the right main bronchus just below the orifice of the right upper lobe bronchus. As on the left side, the upper pulmonary vein is closely associated with the artery. Adjacent to the right lower lobe bronchus and especially its medial branch is the right lower pulmonary vein (fig. 1).

It is evident that in examining the lung, in removing foreign bodies and especially in taking a specimen of a cancer for biopsy the relations of the pul-

monary vessels to the various bronchi are of considerable importance, and the course of each of these vessels should be constantly kept in mind by the endoscopist.

As is well known, the cartilaginous rings of the trachea and main bronchi are deficient on the dorsal surface, so that this wall protrudes more easily into the lumen. On dissection the hilar glands, especially those at the bifurcation, are usually noted and explain how changes in the contour of the carina and the bronchi occur when the glands become much enlarged.

AIR-INFLATED HUMAN LUNGS FOR TEACHING AND DEMONSTRATION

Human lungs may be inflated with air and used for teaching and demonstration purposes. Lungs freshly removed at autopsy and lungs from cadavers may be used. In either case it is essential that the lungs be free of disease in order that they may be completely filled with air. The lungs are removed intact with the trachea. The latter is then attached to rubber tubing which is connected to

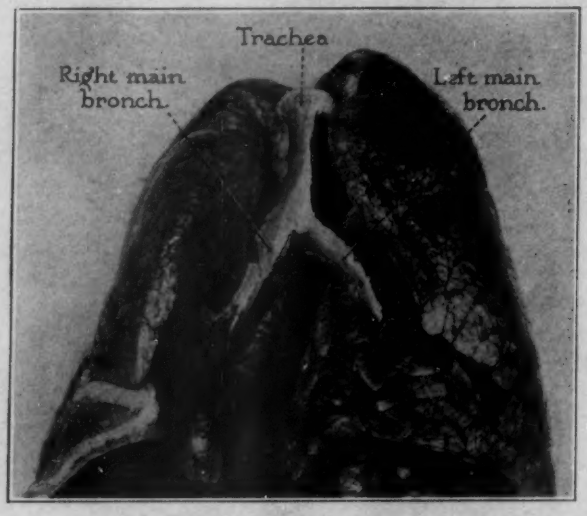


Fig. 4.—Photograph showing the anteroposterior view of human lungs prepared by a technic employing compressed air inflation.

the compressed air line. When the compressed air is slowly turned on, the lungs expand. The pressure is increased until the expansion is full and complete. The lungs are left this way, with the pressure just strong enough to keep them from showing any collapse. In lungs from cadavers the pressure is kept up continuously for about a week, while in lungs freshly removed at autopsy three days is usually sufficient. If the airline contains water at times, a simple bottle trap should be interpolated so as to remove it. It is advisable to change the position of the lungs from time to time, thus also affording an opportunity to inspect all portions of the lungs. The pressure can be increased or decreased at any time when the appearance of the lungs so indicates.

When the lungs are dry and fixed, it will be found that the air pressure may be turned off and there will be relatively little contraction of the lung tissue. Whatever shrinkage there is of the lungs is not sufficient to be noticeable. From this point on the lungs may be treated variously, depending on the use to which they are to be put. In some instances the lungs may be left just as they came off the airline. In the lungs shown in figures 4 and 5 the trachea and bronchi

were dissected open for about one-half their circumference. The remaining portions of the trachea and bronchi were then enameled in individual colors, and the external borders of the lobes were striped in the corresponding colors. Thus the upper lobe bronchi and lobe borders are in red; the right middle lobe bronchi, in yellow; the trachea, the main bronchi and the lower lobes, in white. The surfaces of these lungs, especially those of the lungs from cadavers, can be laquered with one, two, three or more coats. The laquering, if to be done, should precede the enameling. While the lungs shown in figures 4 and 5 are attached to the trachea, it is advantageous in some specimens to separate the lungs and dissect the secondary bronchi more thoroughly, or to dissect the lung in any way desired.

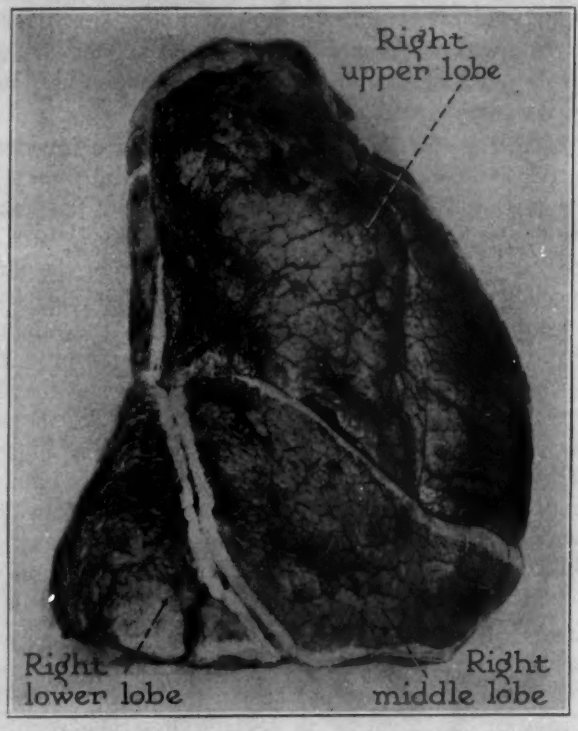


Fig. 5.—Photograph showing the right lateral view of the preparation shown in figure 4.

In my experience lungs prepared in the manner described will remain without appreciable change for an indefinite time, at least for several years. It has been noted that fresh lungs as obtained at autopsy have practically no odor even when unlaquered. However, they are more friable and stand handling less well than lungs from a cadaver. The latter tend to have some odor, but this is not objectionable, especially when they are being demonstrated to persons medically trained or in a fairly well ventilated room. I believe that by using such preparations it is possible to show the bronchoscopic relations better than by any other method. Such preparations also make it possible for the internist, the thoracic surgeon, the roentgenologist and the endoscopist to have an actual lung before them in working out any case at point.

George W. Bartelmez, Ph.D., of the department of anatomy of the University of Chicago, contributed suggestions in regard to the technic of lung inflation.

30 North Michigan Boulevard.

FUNCTIONAL ANATOMY OF THE EUSTACHIAN TUBE

CLEVELAND S. SIMKINS, PH.D.
Professor of Anatomy, Creighton University School of Medicine
OMAHA

Numerous investigators have contributed to the knowledge physicians have of the anatomy, physiology, development and pathology of the tube which Eustachius described in 1563 and which has since borne his name. That the function of the tube is to equalize the pressure of air in the tympanum all students agree, but there is no such universal agreement as to how the equalization is accomplished and just how the tube is opened and closed.

Some of the explanations of the way in which the tube functions are the following: (1) The tube is opened by the tensor veli palatini muscle and closed by the levator; (2) it is closed by the tensor and opened by the levator; (3) it is opened by both the tensor and the levator; (4) it is closed by both the tensor and the levator, assisted, perhaps, by the superior constrictor of the fauces; (5) it is opened by the faucial muscles assisted by the salpingopharyngeus muscle; (6) it is opened by the levator muscle only, possibly assisted by the salpingopharyngeus; (7) it is closed by the levator only and opened by the tensor. McMyn [1] stated that the tube is opened mainly by the tensor, the levator, and the salpingopharyngeus muscle and the upper fibers of the constrictor muscle, the two last-named muscles acting as synergists. He contended that the closing of the tube is passive and is aided by the elasticity of the cartilage. In his opinion the tube is subject to wide individual variation, in some persons being always open and in other persons always closed, in some instances by mucous plugs. The contradictions and the lack of uniformity of opinion concerning the way in which the tube functions lead to the conclusion that the modus operandi has yet to be ascertained.

Perlman [2] stated that after resection of the gasserian ganglion in which the motor root was injured the abnormal patency which followed pointed to the tonic effect of the extrinsic muscles of the eustachian tube supplied by the fifth nerve (the tensor veli palatini) as a factor in keeping the tube normally closed and offering definite resistance to its opening. This circumstance also emphasized the need for reexamining the concepts pertaining to the functions of all muscles acting on the eustachian tube.

With these points in mind the auditory tube has been reexamined in the cadaver and in the living body (by means of the nasopharyngoscope), and this reexamination has yielded information leading to concepts perhaps hitherto overlooked.

RESULTS FROM DISSECTIONS

The tube may be divided into four sections: membranous, cartilaginous and osseous portions and the isthmus. The membranous section is short, occupying from 4 to 6 mm. of the tube and constituting the torus tubarius.

1. McMyn, J. K.: Anatomy of the Salpingopharyngeus Muscle, J. Laryng. & Otol. **55**: 1-26, 1940.

2. Perlman, H. B.: The Eustachian Tube: Abnormal Patency and Physiologic State, Arch. Otolaryng. **30**:212-238 (Aug.) 1939.

Torus Tubarius.—According to Schwartzbart,[3] the opening through the torus tubarius in the fetus and the infant is triangular, being so revealed in cross sections of the tube at right angles to the long axis of the lumen. One angle is placed medioposteriorly, and the other two angles run together into a half-moon–shaped opening laterally and under the concave portion of the lumen, the mucosa of which is thrown into longitudinal folds.

McMyn [1] stated that the salpingopharyngeus muscle descends along the posteromedial fold of the torus tubarius and continues as low as the superior constrictor pharyngis muscle, often to a level equal with that of the thyroid cartilage of the voice box, with which, however, it never actually forms contact. With this observation I am in agreement. But that the ostium is filled with mucus which acts as a plug to close up the tube is not in accord with observations made on the living torus tubarius.

Two distinct types of torus tubarius may be observed, that in the cadaver and that in the living body. The main differences are in the prominence of the folds and in the depth of the ostium. In the living torus the aperture is scarcely dis-

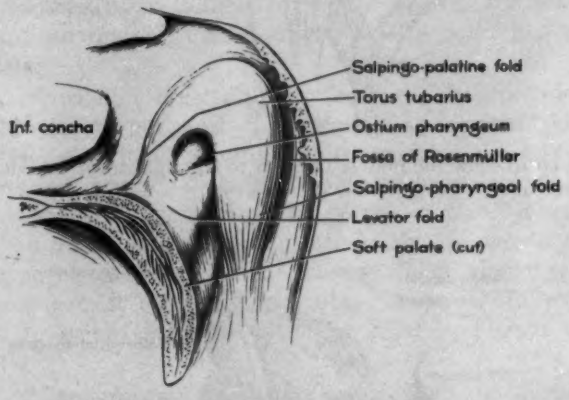


Fig. 1.—Medial side of the right torus tubarius and the pharyngeal ostium of the auditory tube as it lies covered with mucous membrane in the cadaver.

cernible and is always covered by a mucous blanket, which gives the whole the appearance of being freshly varnished and imparts an orange cast. In the cadaver the folds are high (fig. 1) and the aperture is sunken, the surrounding shrunken folds giving no hint that they once were capable of movement.

Behind the torus is the fossa of Rosenmüller, wherein the greatest activity takes place in the pharynx when the soft palate is closed or relaxed and which, even in the living subject, varies considerably in depth. When the bar of Passavant is prominently constricted, as in the closing of the pharyngeal isthmus, the fossa of Rosemüller is transformed into a digital fossa, unlike the linear one observed in the cadaver (Townshend [4]).

Three structures enter into the formation of the torus tubarius: the nasal mucous membrane, covered with the mucous blanket, which is kept moving by the effective strokes of the cilia toward the throat, the muscle fibers, and the blunt end of the tubal cartilage, which protrudes prominently into the nasopharynx and

3. Schwartzbart, A.: Die fetale und postfetale Tuba Eustachii: Anatomisch-histologische Untersuchungen, Cracow, Imprimerie de l'Université, 1938, pp. 1-160.

4. Townshend, P. H.: Formation of Passavant's Bar, J. Laryng. & Otol. **55**:154-165, 1940.

after death is responsible for the shape of the entire bulla. The covering of the torus is nasal mucosa, under which are to be found two bundles of muscle fibers, both derived from the pharyngeal wall (figs. 1, 2 and 3). The figures cited were drawn from the medial inspection of the right cadaveric tube, in which the shape of the ostium has contracted enough to depart markedly from its form in the living body. As revealed by the nasopharyngoscope the torus is not so pronounced and the ostium is scarcely discernible until the person swallows or pronounces the letters M or N or the word nine, when the mucous blanket, sweeping over the

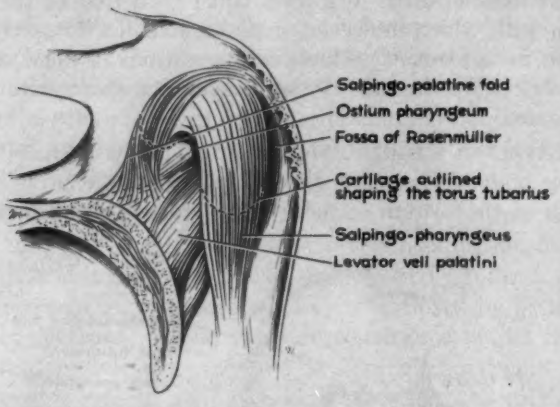


Fig. 2.—Dissection to show the muscle fasciculi of the right torus tubarius after the removal of the mucous membrane. (Drawn from the cadaver.)

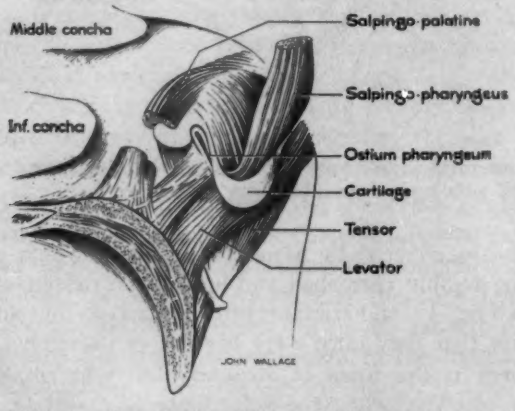


Fig. 3.—Dissection of the muscles of the right eustachian tube and their relation to the cartilage.

opening, is seen to break in a slitlike opening which bows upward in its center until it assumes the form of a definite crescent, one sharp limb being anterior and low, the other posterior and low, the convexity arching upward over the roll formed by the levator muscle. This slit endures for two fifths of a second in the act of swallowing and then the edges of the mucous blanket reunite and sweep on with the cilia toward the throat. All acts of swallowing do not cleft the blanket, and not all movements of elevation of the soft palate cause the blanket to break. But with every act in which the pharynx is elevated or the soft palate is raised tension on the folds of the torus is reduced.

The anterior fold of the torus is made by a few delicate fibers of muscle which I have designated the salpingopalatine fold, and I have referred to the muscle as the salpingopalatine muscle. The fine fasciculi arise from the superior lateral border of the cartilage and arch forward and downward to lose themselves in the soft palate near the junction of the two parts, hard and soft. The posterior welt of the torus is made by the salpingopharyngeus muscle, which arises from the medial superior border of the cartilage and arches downward to play out into the lateral wall of the pharynx in close association with the palatopharyngeus muscle. Its insertion into the lateral pharyngeal wall is variable, and the lower fibers may often be closely united with the palatopharyngeus muscle. In the bottom of the ostium is a prominent roll made by the levator, which is always separated from the cartilage and the tubal tissue by a layer of fat in a loose connective tissue stratum.

In figure 3 the two tubarius muscles of the torus tubarius, the salpingopalatinus and the salpingopharyngeus, have been sectioned and reflected to show their relation to the levator and the tensor muscle of the soft palate. In straddling the levator the two muscles (the salpingopalatinus and the salpingopharyngeus) form a definite means for pinching the soft tubal membrane against the levator and thereby converting the ostium into a "flutter valve," whose operation will be discussed more in detail later.

The tensor veli palatini muscle arises from the lateral side of the hooked cartilage in feeble strands, but most of the muscle arises from the bone on top of the hook and adjacent to it, in a continuous sheet from the torus tubarius as far distally as the osseous junction. As the sheet of fibers descend along the lateral hook they converge on the hamulus of the pterygoid plate, where the tendon, at right angles to the descending fibers, crosses in the groove to reach the palatal aponeurosis. At no place are the muscle fibers in direct contact with the cartilage; they are separated from it by the lateral tubal tissue. Distally the fibers of the levator play out in origin from the scaphoid fossa and from the spine of the sphenoid (fig. 4).

The levator veli palatini muscle has its origin from the medial surface of the petrous portion of the temporal bone below the junction of the osseous and the cartilaginous portion of the tube. No fibers arise directly from the medial roll of the cartilage. Everywhere there is a layer, several millimeters thick, filled with fat and loose connective tissue, which intervenes between the muscle and the cartilage. The fasciculi form a wide band which is inserted into the sides of the palatal aponeurosis. The tensor muscle is supplied by nerve fibers from the fifth nerve, by way of the otic ganglion, and by fibers from the pharyngeal plexus, perhaps mostly from the ninth nerve.

The levator muscle is supplied by the seventh nerve. According to Moritz (1938),[5] the fibers of the levator are incorporated into the intermediate division of the facial nerve. The fibers course through the chorda tympani to the otic ganglion and from there are carried by the nervus sphenoidalis internus to the nervus petrosus superficialis major and by the Vidian nerve to the sphenopalatine ganglion. From the sphenopalatine ganglion fibers are delivered through the posterior palatine nerves to the levator veli palatini muscle. The tensor is supplied by the ninth nerve by way of the pharyngeal plexus and the fifth nerve through the otic ganglion (Moritz[5]).

5. Moritz, W.: Funktion und Innervation der Muskulatur der weichen Gaumens, Ztschr. f. Anat. u. Entwcklngsgesch. **109**:197-206, 1938.

Cartilaginous Portion.—The cartilage is grooved laterally and rounded medially. It is about 14 mm. in length and 8 to 10 mm. wide near the ostium, but this size tapers gradually to a mere shelf as it joins the osseous part of the tube. It is then club shaped, gouged and indented on its lateral side. At its junction with the osseous portion of the tube it rides on top of the lumen and is continuous with that thin shelf of bone which separates the lumen of the isthmus from the tube containing the tensor tympani muscle (fig. 4).

The cartilage may be in a single piece, or it may be cleft into several pieces which are inserted along the course of the tube. It must be pointed out that figure 4 is a diagram in which use is made schematically of the generalized features of the cartilage. Laterally the single piece of cartilage hooks over the lumen into the bone of the pharynx. At the pharyngeal ostium the cartilage is covered by the salpingopharyngeus muscle, which ties the club end against the levator bulla. So the action of the salpingopharyngeus is governed slightly by the levator muscle.

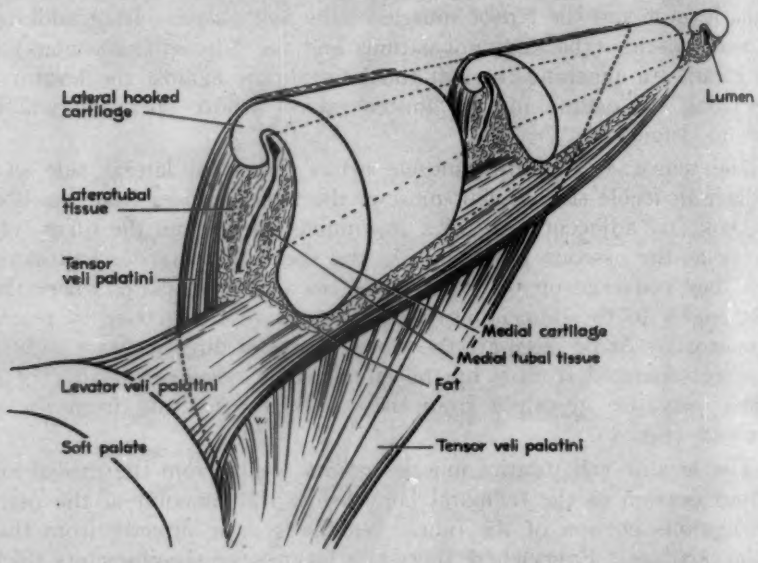


Fig. 4.—Diagram of the cartilage and the lumen of the eustachian tube and the related muscles from the right side of the body. The relation of the levator veli palatini muscle to the cartilage is considerably out of proportion, and its origin from the petrous portion of the temporal bone is not shown.

The lateral roll of the cartilage which forms the tubal hook is smaller from the proximal to the distal end than is the medial roll, which may reach a length of 10 or 12 mm.; but the lateral roll retains its relation to the lumen laterally through its extent, while the medial roll diminishes distally (toward the ear) in such a way that near the isthmus, or the union of the cartilaginous with the osseous portion, the medial roll has so played out that it rides high over the lumen and actually, at the junction, is larger laterally than medially. The arrangement of the cartilage with reference to the lumen serves a definite function. In the first place, the hook serves as a hinge along which the cartilage may be raised or lowered in one piece, which assures equalization in tension or release of tension all along the tube and thus permits uniform relaxation throughout the lumen, so that local areas or bands or segments of the tube will not interfere with adjustments of air pressure between the tympanum and the nasopharynx.

Tubal Tissue.—There are two rolls of paratubal tissue, the lateral and the medial. The laterotubal tissue is firm and spongelike. It is placed medial to the tensor muscle and lateral to the epithelium, in such a way that it makes a distinct longitudinal roll against the lateral wall of the lumen. The medial tubal tissue is not so firm; it contains many glands and is arranged along the medial side of the tubal slit, above the levator muscle yet lateral to the main medial cartilaginous plate.

Lumen.—At the pharyngeal ostium the lumen is normally collapsed and its walls are folded parallel with its long axis; throughout the cartilaginous portion of the tube it is cushioned in between the lateral and the medial tubal tissue.

The osseous portion of the tube, from 12 to 14 mm. in length, transmits the isthmus. Muscles and cartilage play out, and the lumen assumes a more truly tubular appearance. It expands into the hypotympanum, below and in front of the tympanic cavity proper. The osseous portion has not been inspected with care, because it was believed to have little influence on the function of the tube as a whole. The lumen through the osseous portion is normally open and varies from 3 to 6 mm. in diameter. Being held rigidly open by the bony walls, the lumen cannot be closed or opened passively, permitting no deterrent from and no aid to equalization in changes of pressure. The epithelium of the lumen is a modified nasal type. Cilia are said to be present in the pharyngeal end, which is wide, but these play out about the middle of the cartilaginous portion. Glands, mainly from the medial side, open into the epithelium all along its route. Outside the epithelium lymphoid tissue abounds in varying amounts according to age and position or place from which inspection is directed.

The lumen is shaped like a gouge, wide at the pharyngeal ostium and narrowing as it approaches the osseous portion. Its upper and lateral angle lies under the cartilaginous hook and is lateral to the great cartilaginous bulla. Near the center of the cartilaginous portion the lumen lies almost directly under the arch of the hook, and at the osseous junction it lies for the most part medial to the lateral arm of the hook, which at that place is larger than the medial arm. Through the isthmus the lumen averages 4.5 mm. in height. Its extremes, given by Schwartzbart,[3] range from 3 to 6 mm. in height and from 2 to 5 mm. in width. In infants it may be 2 to 4 mm. high and 0.5 to 2 mm. wide.

From the ostium pharyngeum to the ostium tympanum (hypotympanum) the slitlike lumen compressed into a collapsed cylinder like a gouge is spiraled into a lazy S-shaped turn lying on its side. The first lazy twist of the spiral is a lateral turn of the upper, or cranial, margin of the lumen under the hook just distal to the ostium pharyngeum. The bending of the slitlike channel at the center of the cartilaginous part of the tube is neutralized, while the tympanic part of the S-shaped spiral lumen bends medially. This bend is opposite the spiral-like twist of the cartilage, which turns toward the medial side of the lumen near the pharynx and away from it toward the lateral side near the isthmus. The whole cartilaginous arch is thus curved on top and fixed, while the under surface remains straight and free, except for the indirect relationship it bears to the levator veli palatini muscle. The attachment of the hook to the lateral bony wall does not strictly parallel the lumen, because the cartilage is high proximally (near the pharynx) and low distally (near the tympanic ostium). One must think of the cartilage and the lumen as making a lazy twist from the torus tubarius to the osseous junction, which has a definite effect on the manner in which the tube is closed and opened.

MODUS OPERANDI OF THE EUSTACHIAN TUBE

Schwartzbart[8] has convincingly contended that the form of the delicate lumen cannot be accurately determined by the method of injecting molten metal under high pressure into the tube, because it is surrounded by paratubal tissue, the lateral roll being firm and relatively unyielding and the medial being soft, and is relatively easily displaced. Hence, the reliable method is reconstruction of a series of cross sections, which even in the cadaver depart from the normal pattern in the contraction and the shrinkage which inevitably follow rigor mortis and hardening in solution of formaldehyde. But, from an inspection of the torus tubarius in the living body and a manipulation of the soft parts in fresh specimens the manner of operation may be explained as follows:

The torus tubarius with its two straddling muscles, the salpingopalatinus and the salpingopharyngeus, constitutes a "flutter valve" which is released and is tightened downward on the levator. Every act which tends to elevate the soft palate, to pull up the pharynx or to reduce tension on Passavant's bar releases the tension on the valve. Hence all movements which increase tension on the lips of the "flutter valve" close the tube; these include inspiratory effort, reclining posture, bending of the head forward on the chest, attempts to pull the Adam's apple out of the collar and reflex contraction of the muscles by strong pressure on the tragus. Movements, then, which tend to release tension and, therefore, to make opening of the valve easy are acts, complete or incomplete, such as swallowing, elevating the soft palate as in pronouncing the letters M and N and the word nine, coughing, belching, sneezing, blowing the nose, holding the nasopharynx closed and yawning. Other reflex actions all tend to reduce tension on the lips of the valve and to make opening the tube easy.

The release of tension of the valve is only the first step in operating the lumen as a whole. Here the most important structure is the cartilage, which, as we have shown, is a gouge-shaped club, which is free below and medially and is fastened above and laterally by a hook which is a typical hinge. With every effort to depress the pharynx, the larynx and the soft palate the levator is slackened, allowing the salpingopharyngeus muscle to pull the cartilage downward and laterally in a spiral manner; with elevation of the soft palate and consequent contraction of the levator muscle the medial paratubal tissue is pushed upward and under the cartilage so as to unwind the spiral upward and laterally. This movement takes place along the whole length of the cartilaginous portion of the tube synchronously, so that uniform adjustments may be made for the passage of air along the entire length of the tube, the direction of the passage being governed by inequalities in pressure. If the pressure is greater in the tympanum air will move toward the pharynx, and if it is lower in the cavity of the drum air will move in that direction. The tensor muscle of the soft palate, by these same movements, pushes the laterotubal tissue into the lateral wall against the lumen and therefore aids in keeping the lumen collapsed. Both the tensor and the levator are passively acting muscles, when their action on the tube is considered. The levator assists in opening the tube only by allowing the spiraled cartilage and the lumen to untwist. The tensor is a passive closer of the lumen by its compression of the laterotubal tissue, which it presses into the lateral wall of epithelium, converting the lumen into a spiral gouge-shaped slit.

Two parts are to be recognized which operate harmoniously to keep the tube closed in normal activity, a valve and a lumen. Active pull of two muscles in contraction, the salpingopalatinus and the salpingopharyngeus, against the roll of the levator pinches the valve closed. Opening is accompanied by release of

the tension on the lumen, and this is accomplished by the levator. The tensor is the antagonist of the levator on the palate; it depresses the soft velum while the levator pulls it upward and to the rear. When the tensor contracts, its secondary action is to push inward the laterotubal tissue; when the levator contracts, it pushes up the cartilage and allows the spiral lumen to untwist. Therefore air pressures of higher value will move to compartments of lower pressure. The tube is opened passively by inequalities in air pressure and is assisted in an action which can only be secondary, by the levator. Closure is aided by the fact that the tensor secondarily pushes the laterotubal tissue into the lumen.

The antagonistic action of the tensor and the levator on the lumen affords an effective means for pumping fluids out of the tube which would otherwise collect there. Air movements through the tube are always slight and are through a slit which is never more than a millimeter or a fraction of a millimeter open. Inequalities in air pressure open the tube; the closure is brought about by an active contracture of muscle in which the valve is pinched shut. Pumping action all along the lumen is a secondary action, an effect of the action of the antagonists, the tensor and the levator.

Any force, such as the insertion of a catheter into the tubarius, which tends to pry up the spiraled cartilage at its club-shaped end loosens tension all the way along the cartilaginous wall of the lumen and assures uniform adjustment of the entire gouge so that the air may pass through unhindered by local constrictions.

In infants the torus is placed low with reference to the inferior concha. As the pharynx elongates in maturity the torus tubarius comes to occupy a higher and higher level. If it does not ascend high enough or the pharynx is not pulled low enough, tension on the "flutter valve" is always inadequate and the tube opens at such low pressures that the patient hears his own breath sounds. It is true, also, that the valve may be placed so high that it opens with ever increasing difficulty. Both conditions lead to grave impairment of hearing. Between these two extremes of an abnormally patent tube and an abnormally closed tube, varying degrees of tension on the valve exist. These values vary not only in different persons but in the same person at different times, because of automatic nervous impulses, barometric pressure, neurologic state and pathologic conditions of various kinds.

Mucus has never been observed to form a plug anywhere about the ostium and the contention of McMyn that such mucus plugs exist which close the tube has at no time been borne out in the investigation of the living torus tubarius. Over the ostium in the living condition the mucus forms a "blanket," which is continuous, is thin and is kept moving by the cilia toward the throat. This blanket is seen to cleave immediately over the ostium as the first step in the opening of the ostium. Release of muscle tension allows the blanket to break, and constriction again draws the torn edges of the blanket together and enables them to reunite and sweep on as they did before the rupture.

SUMMARY

The cartilage and the lumen of the eustachian tube form a spiral lazy "S" from the pharynx to the osseous junction which keeps the lumen collapsed against the laterotubal tissue and hence closed. The gougelike lumen untwists on release of muscle tension.

The torus tubarius is a "flutter valve" which is actively closed by the tension of the salpingopalatinus and the salpingopharyngeus muscle.

The levator veli palatini muscle assists in equalizing adjustments all along the lumen by elevating the spiral of the cartilage and the lumen, a movement which is passive.

The "flutter valve" is closed actively by muscle tension but opened passively by inequalities in air pressure, either on the pharyngeal side (nasopharynx) or on the tympanic side of the shutter.

The function of the cartilage is to assure uniform adjustment in maintaining tension, or in releasing tension all along the lumen of the cartilaginous portion of the tube.

The tensor veli palatini muscle assists passively in compressing the laterotubal tissue into the lateral wall of the lumen, thereby holding it collapsed.

The antagonistic action of the tensor (passive collapser of the lumen) and the levator (passive untwister of the spiral) produces a squeezing, or pumping, action on the lumen and aids in the expulsion of fluid from it.

The Creighton University School of Medicine.

LUDWIG'S ANGINA

AN ANATOMIC STUDY OF THE ROLE OF THE LOWER MOLAR TEETH IN ITS PATHOGENESIS

KURT TSCHIASSNY, M.D.
CINCINNATI

Dr. Karl Friedrich Wilhelm von Ludwig [1] deserves a permanent place in medical history because he recognized the disease now called by his name as a distinct morbid entity characterized by anatomic, pathologic and clinical signs peculiar to it and differentiating it from lesions closely resembling it. Of course, his assumption that he had met with a new kind of inflammation of the neck of an epidemic character was erroneous.

HISTORY

The disease had been known since classical antiquity and had been mentioned by Hippocrates, Galen, Caelius, Aretæus, Paulus Aegineta, Aurelianus, Severinus and others, and it appears in the later literature under various names. For example, it was classified as "cynanche," "carbunculus gangraenosus," "morbus strangulatorius," "esquinancia gangraenosa," and "angina maligna." The Spaniards called it "garotillo" (after "garote," a loop used by hangmen) and the French "l'esquinancie inflammatoire gangreneuse" (Deberge, 1758) or "phlegmon large du cou" (Dupuytren, 1833). The occurrence of the disease had been observed and described by Colden in New York (1735) and by Cholmer (1770) in South Carolina. Dr. Thomas Kirkland (1786) reported a case of "angina externa" in which he saved the life of the patient by an early deep median incision, while General Washington succumbed to a hemorrhage caused by "cynanche trachealis."

A comprehensive history of the disease was presented by Parker,[2] in 1879, and by Muckleston,[3] in 1928. Many of the data had already been mentioned by Heim,[4] a contemporary of Ludwig in questioning Ludwig's assumption that the disease was new.

Ludwig's Description.—These facts do not detract from Ludwig's merit. His classic article was a masterpiece of exact observation and precise description with an almost intuitive recognition of all the significant factors and characteristic details. "The short, clear and comprehensive account which Ludwig gave of it, has been surpassed by no later description of the disease" (Ashhurst [5]).

From the Department of Otolaryngology, University of Cincinnati College of Medicine.

This paper was read at the meeting of the Cincinnati Otolaryngology Society on March 23, 1943.

1. von Ludwig, F. W.: Eine neue Art von Halsentzuendung, Med. Cor.-Bl. d. württemb. ärztl. Ver. **6**:21, 1836.

2. Parker, R. W.: Remarks on Cellulitis of the Neck (Angina Ludovici), Lancet **2**:570 and 607, 1879.

3. Muckleston, H. S.: Angina Ludovici and Kindred Affections: Historical and Clinical Study, Ann. Otol., Rhin. & Laryng. **37**:711, 1928.

4. Heim, L.: Zur Geschichte der Metaphlogose des Zellgewebes am Halse, Schmidt's Jahrb. **15**:25, 1837.

5. Ashhurst, A. P. C.: Ludwig's Angina: Primary Focus About the Teeth, Arch. Surg. **18**:2047 (May) 1929.

According to Ludwig's observations at the bedside and at the autopsy table, the observations to be emphasized as characteristic and the most important are:

1. Ludwig's angina is a rapidly spreading gangrenous cellulitis.
2. It originates in the region of the submaxillary gland (i. e., spatium submaxillare or submandibulare).
3. The extension evidently occurs by continuity and not by the lymph channels.
4. It shows no special tendency to form abscesses.
5. There is plainly no involvement of the submaxillary glands and lymph nodes.

Dissenting Views.—However, despite Ludwig's exactness, some of those sharply defined characteristics "by which this disease differentiates itself from those other diseases whose outward appearance is somewhat akin "(Burke's [6] translation of Ludwig's article) have been frequently overlooked by various later observers. Roser [7] and others assumed that Ludwig's angina is a specific disease of the submaxillary salivary gland—a sort of submaxillary mumps. This idea, however, has been definitely abandoned.

Von Thaden,[8] Poulsen,[9] Stengel,[10] Thomas [11] and others considered the disease to be primarily a submaxillary lymphadenitis, even suggesting the term "submaxillary bubo" (von Thaden).

Furthermore, several French writers (Délorme, Leterrier, August, DeBovis and others) [11] considered the disease as sublingual cellulitis. The term Ludwig's angina was even regarded as synonymous with sublingual phlegmon. There was a strong tendency to drop the term Ludwig's angina altogether, since the disease was not believed to deserve a special place in surgical pathology. This idea persisted intermittently down to the last decades (Finder,[12] 1925).

Pseudo Angina Ludovici.—At the beginning of the century, some useful work was done in order to restore the picture of the disease to its original distinctness. Davis [13] emphasized the fact that Ludwig's angina spreads along connective tissues by direct continuity, is not transmitted by lymph channels and does not affect lymph nodes. This was soon demonstrated histologically, by Coplin,[14] who was able to show that there is no infiltration of lymph nodes in Ludwig's angina.

On the other hand, Trautmann [15] mentioned a "secondary" type of phlegmon of the neck, which is actually caused by inflammation of lymph nodes consequent on infection in their drainage area. "This, of course, is not the same as the type

6. Burke, J.: Angina Ludovici: Translation, Together with a Biography of Wilhelm Frederick von Ludwig, Bull. Hist. Med. **7**:1115, 1939.

7. Roser, W.: Die Ludwig'sche Angina, Deutsche med. Wchnschr. **9**:153, 1883.

8. von Thaden, cited by Thomas.[11]

9. Poulsen, K.: Ueber Abscesse am Halse, Deutsche Ztschr. f. Chir. **37**:55, 1893.

10. Stengel, A.: Textbook of Pathology, ed. 3, Philadelphia, W. B. Saunders & Co., 1900, p. 493.

11. Thomas, T. T.: Ludwig's Angina: An Anatomical, Clinical and Statistical Study, Ann. Surg. **48**:161 and 335, 1908.

12. Finder, G., in Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohren-heilkunde, Berlin, Julius Springer, 1928, vol. 3, p. 365.

13. Davis, G. G.: Acute Septic Infections of the Throat and Neck: Ludwig's Angina, Ann. Surg. **44**:175, 1906.

14. Coplin, in discussion on Thomas.[11]

15. Trautmann, G.: Ueber eine dentale, unter dem Bilde der Angina Ludovici auftretende und mit Retropharyngealabszess kombinierte Halsphlegmone, München. med. Wchnschr. **55**: 627, 1908.

described by Ludwig." A similar differentiation was made by Van Wagenen and Costello,[16] based on their careful studies and observations of 16 cases. They even made a terminologic distinction between these two types using the terms "primary" and "secondary." Axhausen[17] and Wassmund[18] made a distinction between a phlegmonous and an "abscess-forming" type. They pointed to the fact that in the latter type the infectious agent passes by lymph channels throughout the submaxillary lymph nodes, whereas in the phlegmonous type it immediately spreads from the bone into the tissue of the floor of the mouth. None of these investigators observed any mortality with the so-called secondary or "abscess-forming" type.

It becomes clear that these diseases cannot be regarded as forms of Ludwig's angina. They actually are forms of suppurative submaxillary lymphadenitis enlarging in the direction of the mouth, a comparatively benign imitation of Ludwig's angina. In order to emphasize the outward similarity without identifying the disease with that described by Ludwig, I occasionally used in discussion the term "pseudo-angina Ludivici" or "pseudo-Ludwig's" to designate this clinical entity.[19]

Distinction from Sublingual Cellulitis.—Thomas[11] deserves the merit of having disapproved of the erroneous assumption advanced by the French school that Ludwig's angina is merely sublingual cellulitis. After a profound study of 104 cases described in the literature and 2 cases of his own, as well as anatomic studies, he was able to show that (1) Ludwig's angina starts almost regularly in the submaxillary space, (2) it only secondarily involves the sublingual space, and (3) the extension follows the route of the submaxillary salivary gland which turns around the posterior border of the mylohyoid muscle, passing through the gap in the buccopharyngeal wall. Isolated cellulitis either of the submaxillary space or of the sublingual one cannot be regarded as Ludwig's angina. *"It is the simultaneous involvement of both that warrants the condition being recognized as a distinct clinical entity"* (Ashhurst[5]).[20]

The importance of the mylohyoid muscle and its fascial layers as a factor determining the course and the character of the disease has been repeatedly pointed out (Piersol,[21] Houser,[22] Grodzinsky,[23] Williams[24] and others). "It acts the same

16. Van Wagenen, W. P., and Costello, C. V.: Sublingual Phlegmon: Primary and Secondary Ludwig's Angina, Ann. Surg. **87**:684, 1928.

17. Axhausen, G.: Zur Klassifikation der eitrigen Entzündungen am Kiefer, Chirurg **1**:459, 1929.

18. Wassmund: Ueber Mundbodenphlegmonen, München. med. Wchnschr. **77**:892, 1930.

19. Hajek occasionally called it "Ludwigoid."

20. These anatomic conditions differentiating Ludwig's angina from sublingual cellulitis are apparently of clinical and perhaps even of therapeutic importance. Since the sublingual space is separated from the oral cavity only by the overlying mucous membrane, an inflammatory process established merely in this space is more apt to empty into the mouth, either spontaneously or by an endoral incision (in contradistinction to a process extending below the diaphragma oris). Houser advocates the endoral incision on the basis of his successful results in several instances. This recommendation is rejected by others. Hauser's cases, however, were instances of isolated sublingual cellulitis as he emphasized. I believe the endoral approach may be justified as the shortest and simplest way to an area of sublingual cellulitis, but for submaxillary-sublingual cellulitis, which practically represents Ludwig's angina, the external approach should be the procedure of choice.

21. Piersol, G. A.: Human Anatomy, Including Structure and Development and Practical Considerations, ed. 5, Philadelphia, J. B. Lippincott Co., 1916.

22. Houser, K. M.: Ludwig's Angina, Arch. Otolaryng. **16**:317 (Sept.) 1932.

23. Grodzinsky, M.: Ludwig's Angina, Surgery **5**:678, 1939.

24. Williams, A. C.: Ludwig's Angina, Surg., Gynec. & Obst. **70**:140, 1940.

as a rubber band," said Novitzky,[25] "preventing escape of gas and fluid into the mouth." "These two structures (the mylohyoid muscle and the deep cervical fascia) therefore tend to confine infection following extractions of lower molars to the deep tissues under the tongue" (Trout [26]).

This muscle is also called "diaphragma oris" because it separates the neck from the floor of the mouth. Its upper surface belongs to the floor of the mouth; its lower surface belongs to the neck. Ludwig [6] described the disease as a "gangrenous induration of the connective tissues of the neck which advances to involve the tissues which cover the small muscles between the larynx and the floor of the mouth." In my opinion, therefore, the term "phlegmon of the floor of the mouth," which is frequently used as synonymous with Ludwig's angina, cannot be considered as entirely adequate to Ludwig's original description of the condition.

Etiology.—Ludwig's idea that the disease is epidemic has been frequently repeated in the last century but is now abandoned. The alleged occurrence of cases of Ludwig's angina in a greater number within a short time may probably be explained as the occurrence of secondary suppurative lymphadenitis in cases of a primarily epidemic disease such as scarlet fever, diphtheria or measles. It is obvious that such infections can be regarded only as pseudo Ludwig's angina. Accordingly, those diseases frequently mentioned as sources of Ludwig's angina cannot be regarded as etiologically related to it. Likewise a number of lesions located in the drainage area of the submaxillary and submental lymph nodes have to be excluded. These are principally lesions about the lips, tongue, cheeks, gums, floor of the mouth, tonsils and pharynx and lesions of the skin such as impetigo, furuncles, erysipelas, lupus erythematosus and otitis externa.

Since Ludwig's angina is originally a submaxillary cellulitis, it can be brought about, as a rule, by an infection which is primarily located below the mylohyoid muscle. The chief sources of infection mentioned in the literature are, in the approximate order of frequency of occurrence, an extraction of molar teeth (apparently the injection for anesthesia around a tooth is sometimes by contamination, a contributing factor), a periapical abscess, a penetrating injury of the floor of the mouth (gunshot wound, stab wound, entrance of a foreign body, horse kick, bite), osteomyelitis and compound fracture of the jaw, otitis media (by way of the digastric muscle, according to Piersol [21]) and an abscess under the thyrohyoid membrane (Blair [27]). Whether a salivary calculus, occasionally mentioned, is apt to produce Ludwig's angina seems to me doubtful. Poulsen,[9] discussing 251 cases of submaxillary inflammation, did not mention it as among the pathogenic factors as far as these have been revealed. The differentiation of an inflammatory swelling caused by an impacted salivary calculus from Ludwig's angina is of course sometimes difficult.[28] Moreover, peritonsillar abscess, although frequently mentioned, seems to me a rather questionable source of Ludwig's angina because the palatoglossal muscle and partly also the styloglossal muscle form a protecting wall in the direction of the sublingual and submaxillary spaces. There may be confusions of peritonsillar abscess with periosteal alveolar abscess of the wisdom tooth as indi-

25. Novitzky, J.: Ludwig's Indurations, Dent. Digest **48**:24, 1942.

26. Trout, H. H.: Ludwig's Angina, Arch. Surg. **41**:532 (Aug.) 1940.

27. Blair, V. P.: Surgery and Disease of the Mouth and Jaws, ed. 1, St. Louis, C. V. Mosby Company, 1912, p. 334.

28. Several years ago I mentioned (in discussion on Wenzel, M.: Ein bemerkenswerter Fall von Speichelstein, Monatschr. f. Ohrenh. **70**:747, 1936) a sign which I believe to be useful for this differential diagnosis. External pressure on the submaxillary region in a case of impacted calculus is followed by a cloudy discharge from the sublingual caruncle. This does not occur, of course, when there is nothing producing a stricture of Wharton's duct.

cated by numerous writers (Trautmann [15]; Burchard [29]; Adrion [30]; Tschiassny [31]; Novitzky [32]).

The predominant role of the teeth among the pathogenic factors of Ludwig's angina was recognized comparatively late. Roser [7] rejected with remarkable vehemence this assumption when it was presented by Bamberger and Niemayer. Parker [2] mentioned periostitis of the jaw among the various etiologic factors but seemed to give it least emphasis. However, at the meeting of the French Surgical Society in Paris in 1892 [5] this condition was extensively discussed, and it has been proved to occur by numerous reports since that time. Ashhurst [5] estimated the frequency of a dental etiologic factor at 65 to 83 per cent; Trout,[26] at 82 per cent; and Wassmund,[18] at 90 per cent. Schlesinger [33] mentioned carious teeth as the only cause he was able to demonstrate, and Novitzky's [32] observations confirmed this. Reports emphasizing the high proportion of cases with a dental etiologic factor have been presented by Bell,[34] Muckleston,[3] Frankenthal,[35] Houser,[22] Ross,[36] Grodzinsky,[23] Price,[37] Taffel and Harvey [38] and many others.

Which teeth are especially involved? Thomas,[11] reviewing the literature down to 1908, made the statement: "So far as reference to special teeth is concerned, they were always molar or wisdom teeth. . . ." Van Wagenen and Costello,[16] having reviewed the literature published since Thomas' review, to 1928, reported the same observation. According to Wassmund, Richard and others, the back molar teeth are the most frequently involved. But several writers (Frankenthal,[35] Muckleston,[3] Grodzinsky,[23] Trout [26] and others) have mentioned the involvement of the premolar teeth also. I was able to find only one report of Ludwig's angina caused by an extraction of a second biscuspid tooth as the only tooth involved. In this case, however, as emphasized by Frankenthal,[35] the dentist was faced with unusual difficulties which had necessitated the trephining of the alveolar process. Trautmann's [15] case was one of sublingual cellulitis following periapical abscess of a first premolar tooth. It is not regarded as a case of genuine Ludwig's angina.

Therefore one is justified in assuming that (according to the observations of numerous writers throughout six decades) the dental focus of Ludwig's angina as far as it has been traced is located almost without exception about the molar teeth. The greatest predilection is shown for the second and third molar teeth, according to some observers.

An explanation of the etiologic predilection for the molar teeth was presented by Moty [39] at the meeting in Paris (1892): From a periapical inflammation following gangrenous pulpitis the pus pierces the alveolus in its thinnest part, which

29. Burchard, H. H.: A Text-Book of Dental Pathology and Therapeutics, ed. 4, Philadelphia, Lea & Febiger, 1912.

30. Adrion, W., in Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohren-heilkunde, Berlin, Julius Springer, 1928, vol. 3, p. 438.

31. Tschiassny, K.: Ueber einen besonderen Fall von "Angina," Wien. klin. Wchnschr. **40**:1003, 1927.

32. Novitzky, J.: Dead Teeth, Inferior Dental Canal Infections and Phlegmons of the Throat, Am. Med. **41**:379, 1935.

33. Schlesinger, A.: Ueber Angina Ludovici, Berl. klin. Wchnschr. **45**:2194, 1908.

34. Bell, J. H.: Ludwig's Angina, Internat. J. Orthodontia **23**:941, 1937.

35. Frankenthal, L.: Angina Ludovici and Its Significance for Practitioner, München. med. Wchnschr. **72**:1150, 1925.

36. Ross, G. G.: Angina Ludovici, Ann. Surg. **33**:720, 1901.

37. Price, J. W., Jr.: Ludwig's Angina: Report of Five Cases, Including One Autopsy, Ann. Surg. **48**:649, 1908.

38. Taffel, M., and Harvey, S. C.: Ludwig's Angina: Analysis of Forty-Five Cases, Surgery **11**:841, 1942.

39. Moty, quoted by Ashhurst.[5]

is on the outer surface for the anterior teeth. It forms in this way a dentoalveolar abscess. If the process is acute (as after ill timed filling of a tooth), there is no time for absorption of the involved alveolus to occur. So the infection enters the dental canal, spreads toward the spine of Spix and makes its way toward the cellular tissue which separates the upper surface of the mylohyoid muscle from the gum. If a back tooth or a wisdom tooth is infected in this way, the outer wall of the mandible is so thick that perforation occurs only on the inner wall, so that the sublingual tissues are infected at once.

This explanation of Moty has been frequently repeated in the literature with some unessential variations up to the present time and is apparently generally accepted (Piersol,[21] Novitzky,[22] Grodzinsky,[23] McCaskey [40] and others). Is it conclusive? There is room for question whether the explanation given by Moty and his followers can be accepted as the indubitable and complete solution of the problem for the following reasons: 1. According to Moty's own words, "the infection . . . spreads toward the cellular tissue which separates the *upper* surface of the mylohyoid muscle from the gum . . . so that the *sublingual* tissues are infected at once." It therefore does not explain the infection of the submaxillary space which is, in fact, the primarily infected region, as has been proved by Thomas.[11]

2. The anatomic conditions on which the explanation of Moty and his followers is based are, in fact, not quite as they are supposed to be by the authors. Transverse dissections of mandibles through the first, second and third molar teeth and the corresponding alveolus as performed by myself show that there is rather an irregularity in the differences of the thickness of the inner wall of the alveolus compared with the outer wall. Only as far as the third molar is concerned do the conditions confirm the statements of Moty. As for the first and second, sometimes there is no marked difference at all, and sometimes the proportions are just reversed, the inner wall being even thicker then the outer one. An example of that is presented in figure 1 (*A, B* and *C*).

Almost the same situation appears in illustrations of a paper by McMillan,[41] which, however, deals with the structures of the alveolar processes from a different point of view.

But even if one intends to assume that the situation in all instances of Ludwig's angina is of the type supposed by Moty, that would explain only why the molar teeth are more apt to transmit an infection toward the lingual side of the alveolus. But the question why just the submaxillary space should become infected in this way by the molar teeth and, moreover, exclusively by the molar teeth remains open.

Several other explanations presented by various authors cannot be accepted, I think, as an exact and entirely satisfactory answer. Thomas [11] simply remarked, in connection with the statement that the molar teeth are the only ones concerned, that they are nearest to the submaxillary region. Somewhat similar but more precise is the explanation given by Trout.[26] Emphasizing that the sockets of the molar teeth are "off center and closer to the inner than to the outer side," he said: "In addition to this anatomic fact it is important to recall that the molar teeth are separated from the underlying fossa of the lower mandible only by a relatively thin partition of the bone." Wassmund [18] expressed the belief that the proximity of the back molar teeth to the posterior border of the mylohyoid muscle makes it probably easier for an infection starting from those teeth to spread simultaneously into both the sublingual and the submaxillary space.

40. McCaskey, C. H.: Ludwig's Angina, Arch. Otolaryng. **36**:467 (Oct.) 1942.
41. McMillan, H. W.: The Structures and Functions of the Alveolar Process, J. Am. Dent. A. **11**:1059, 1924.

ANATOMIC STUDIES

I should like to offer a report on the results of some anatomic studies which I undertook in order to find a solution of this problem. They are probably also of some interest because they reveal an anatomic detail which as far as I know has not been noticed before. They were based on the following considerations:

1. Ludwig's angina starts regularly from a focus below the mylohyoid muscle.
2. This muscle is attached to the mandible at the mylohyoid ridge.
3. Is there a special relation of the roots of the molar teeth to this ridge?

Textbooks of anatomy consulted did not offer any information in this connection. I therefore made examinations as follows: A narrow strip of lead covering the mylohyoid lines was fixed to the jaw on each side. Then roentgenograms were taken of each half separately. Three samples are shown in figure 2. Twenty-three jaws were examined in this way. In the majority of them, of course,

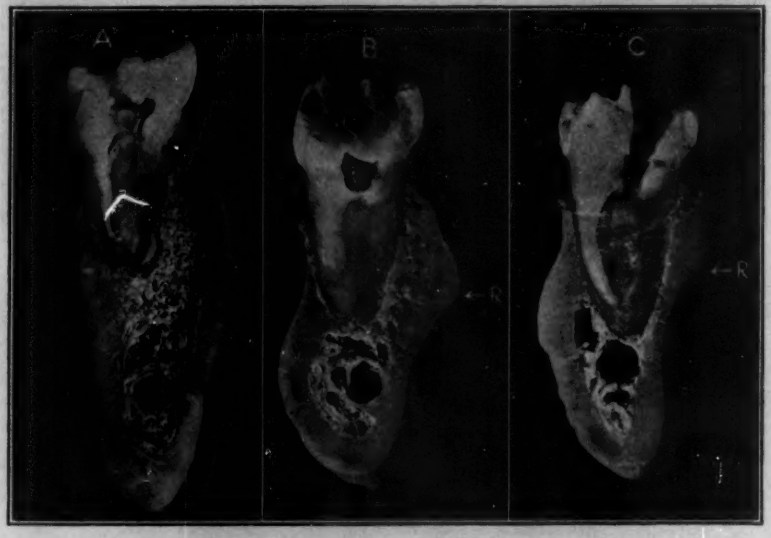


Fig. 1.—Cross sections through the mandible: *A*, first molar tooth; *B*, second molar tooth; *C*, third molar tooth. They demonstrate that the common statement that the lingual plate of the alveolar sockets of the molar teeth is thinner than the buccal plate is not invariably true. Note the difference in the relation of the mylohyoid ridge (*R*) to the apexes of the roots.

many teeth were absent, one specimen even being unilaterally edentulous. Some teeth were markedly pushed upward by alveolar atrophic changes. Altogether, as far as concerns the molar teeth there were present 27 first molar teeth, 32 second molar teeth and 30 third molar teeth.

The relation of the tips of the roots to the mylohyoid ridge, as the roentgenograms made plain, were as shown in table 1. It may be seen in the table that with few exceptions the tips of the roots of the second and third molar teeth reach below or at least as far as the level of the mylohyoid ridge, while a majority of the apexes of the first molar teeth are located above this ridge. As far as the anterior teeth are concerned, their root tips were without exception high above the mylohyoid ridge.

In order to prove that there are no errors in the interpretation of the roentgenograms caused by faulty projections I performed the dissection shown in figure 3. A gap had been cut in the lingual plate of the mandible. The upper border of the

gap represents approximately the level of the mylohyoid ridge. Figure 3 shows the roots of the second and third molar teeth below the mylohyoid ridge. The tips of the first molar tooth are just visible at the level of the ridge. No roots of anterior teeth are visible.

These findings and figures show that under certain circumstances there is a great potentiality for the second and third molar teeth to become the source of submaxillary cellulitis since their roots reach most frequently below or at least to the level of the mylohyoid ridge. The first molar teeth, in contrast with this, are more inclined to transmit an infection into the sublingual space.

The proportion of the respective teeth related to the submaxillary space would be 11 (18 per cent) of the first and 27 (41 per cent) of the second and of the third molar teeth.

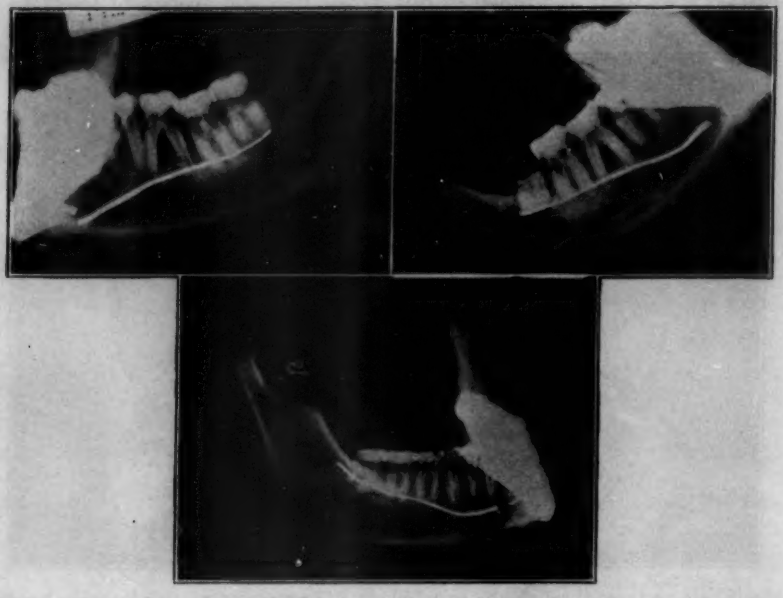


Fig. 2.—Roentgenograms showing the relation of the tips of the roots to the mylohyoid ridge.

TABLE 1.—*Relation of Roots of Molars to Mylohyoid Ridge*

Molar	Teeth	No. with Apex Above the Mylohyoid Ridge	No. with Apex About at the Level of the Mylohyoid Ridge	No. with Apex Markedly Below the Mylohyoid Ridge
I.....	27	16	8	3
II.....	32	5	6	21
III.....	30	3	8	19

These anatomic data further make it plain that occasionally an infected molar tooth or socket not reaching the level of the submaxillary space can become the source of sublingual cellulitis (Houser [22]).

It becomes clear also that the short distance between the border of the alveolar process and the mylohyoid ridge (about 4 to 10 mm. for the second and third

molar teeth) renders it more likely that infectious agents will pass by way of an injection for anesthesia into the submaxillary space.[42]

Comparison with Proportions Compiled in the Literature.—It seemed to me of interest to investigate whether there was a correlation between these anatomic findings and the frequency with which the three groups of molar teeth were etiologically involved in the cases which have been reported. Unfortunately, most of the available literature gives insufficient data by which to determine exactly the particular tooth involved as the primary focus of the infection. For the most part one finds only such remarks as "A molar tooth was extracted" or "Caries of several lower molar teeth was noted." I was able to collect only 24 cases in which the teeth involved were determined. In 6 of them, however, two molar teeth were mentioned. Since it was not possible to determine which of the two was the causative one, I gave both of them the full credit in table 2.

Fig. 3.—A groove on the inner side of the mandible shows the relation of the roots of the teeth to the mylohyoid ridge.

In these 24 cases the first molar tooth was involved six times, the second and the third molar tooth each twelve times. In other words the percentage of the total of 30 molar teeth involved is for the first molar tooth 20 and for the second and the third each, 40.

These clinical figures present a striking correlation with the anatomic data given in foregoing paragraphs. Nevertheless I do not wish to attach too much importance to these figures. They are intended to be taken not as proof but as supporting evidence.

Autopsy Observations Reflecting These Anatomic Statements.—Finally I attempted to see whether these anatomic statements would be paralleled by cor-

42. Ludwig's Angina After Dental Extraction an Accident: Medicolegal Report, J. A. M. A. 95:1045-1046 (Oct. 4) 1930. Price.[37] Davis.[18]

responding observations given in reports of autopsies. However, there are comparatively few reports of autopsies in cases of Ludwig's angina, and most of the reports do not record the condition of the jaw with the exactitude that might be desired.

Ludwig,[16] for example, in reference to the autopsy in a case of his stated: ". . . The periosteum on the inner surface of the jaw was loosened from the bone and discolored. It was at this particular point that the gangrenous ichor first appeared." Unfortunately nothing is mentioned as to the exact location of this point.

Roser [7] made a reference to the report of an autopsy performed by von Recklinghausen. A "foyer purulent" was found starting from an abscess on the root of the fourth back tooth. (This very likely means the second molar tooth.) An autopsy reported by Thomas [11] revealed "on the internal surface of the inferior maxilla a fistula [starting from the second molar tooth] communicating with the

TABLE 2.—*Incidence of Primary Involvement of Each of the Three Molar Teeth*

Author	Cases	Number in Which Infection Was Referred to Given Molar Tooth		
		I	II	III
Ashhurst [5]	1	..	..	1
Blassingame, C. D.: Tr. Am. Laryng., Rhin. & Otol. Soc. 34 : 33, 1928	2	1	1	
Conway, J. F.: J. A. M. A. 118 : 953 (March 21) 1942	1	..	1	
Davis [18]	2	..	1	2
Grodzinsky [23]	3	1	1	3
Hochbaum, W. J.: Laryngoscope 43 : 838, 1933	1	..	..	1
Houser [22] *	1	..	1	
Medicolegal case [42]	1	..	..	1
Muckleston [3]	1	1		
Pratt, G. N.: Illinois M. J. 14 : 462, 1908	1	..	1	
Price [27]	2	1	1	
Ross [36]	2	..	1	2
Thomas [11]	1	..	1	
Van Wagenen and Costello [16]	4	2	2	2
Richard [43]	1	..	1	
Total	24	6	12	12

* Of course, it is not quite certain whether this case is one of Ludwig's angina or one of sublingual cellulitis.

gangrenous focus." Unfortunately, neither of them mentioned any relation of the fistula to the mylohyoid ridge.

The findings of Richard [43] in an autopsy performed by him seem to me to be much more interesting and particularly instructive.

. . . The exact dissection of the neck tracing the gangrenous channels of the tissues leads toward the region of the second lower molar. The periosteum of the mandible is discolored and loosened. After the removal of the periosteum there is found lingually immediately *below the linea mylohyoidea* at the compacta of the bone a hole of the size of a pepperseed. Within this hole the tips of the roots of the molar were distinctly visible. The tooth (second molar) shows a carious cavity.

Figure 4 is a reproduction of two illustrations belonging to Richard's [43] article. The one on the left figure 4 is instructive because it shows the hole below the mylohyoid ridge and the root tips plainly visible within this hole. The one on the right shows the defect at the inner alveolar wall communicating with the carious cavity.

Figure 5 is a photograph of a diagram from the article of Trautmann.[15] It shows (in contrast to Richard's case) a hole in the compacta of the mandible

43. Richard, W.: Zur Genese der Ludwigschen Angina, Frankfurt. Ztschr. f. Path. 45:201, 1933.

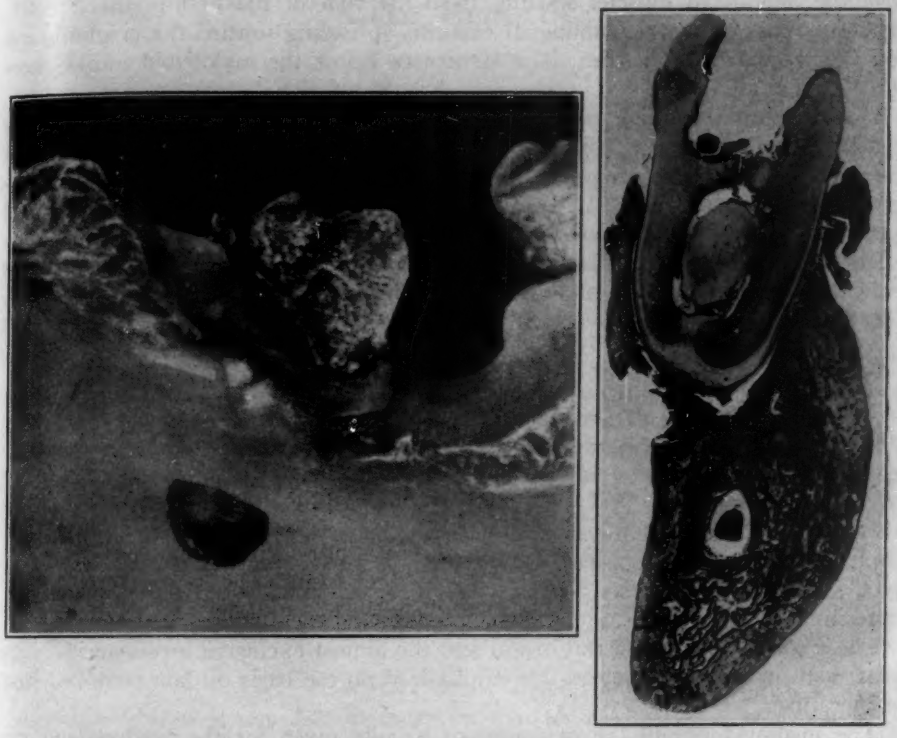


Fig. 4 (Richard's [43] case).—Left, perforation of the inner plate of the mandible caused by caries of the second molar tooth. It is located below the mylohyoid ridge. The root of the molar tooth is visible in the hole. Right, perforation of the inner wall of the mandible at the site of the second molar tooth.

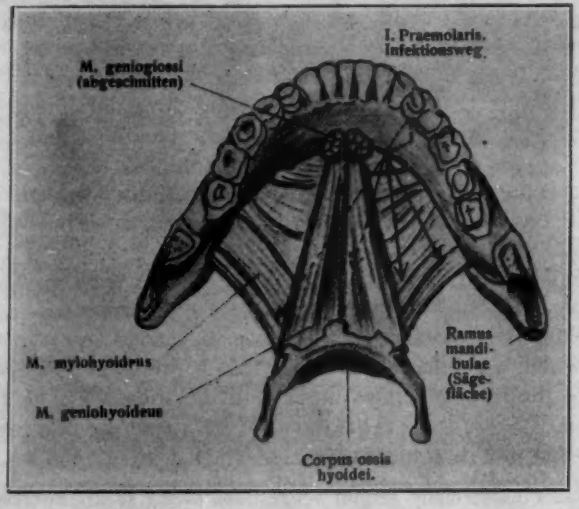


Fig. 5.—Diagram from Trautmann's [15] case. The infection started from the first premolar tooth. Spreading occurred above the mylohyoid muscle.

above the mylohyoid muscle, starting from the root of the first premolar tooth. The result was, of course, sublingual cellulitis spreading toward the epiglottis and even to the contralateral side. The structures below the mylohyoid muscle were not affected at all.

I call attention to these reports because they seem to me in a certain sense a remarkable pathologic and clinical reflection of the anatomic findings as described on a foregoing page. Moreover, they bring out the importance of exactly observing at autopsy in cases of Ludwig's angina the condition of the mandible and especially the state of the molar teeth and the site of a fistula, which apparently sometimes escapes observation unless the swollen and discolored periosteum is removed. A better understanding of the genesis of this disease will contribute to its prophylaxis, diagnosis and therapy.

SUMMARY

A brief review of the history of Ludwig's angina is given. According to Ludwig's description, this entity is characterized by gangrenous cellulitis originating in the submandibular (submaxillary) space and rapidly spreading toward the floor of the mouth.

Neither isolated cellulitis in one of these spaces nor suppurative submaxillary lymphadenitis can be regarded as Ludwig's angina. For the second condition, which sometimes appears as a comparatively benign imitation of Ludwig's angina, the term "pseudo angina Ludovici" or "pseudo Ludwig's angina" is suggested.

The frequency of the dental origin and the almost exclusive involvement of the molar teeth in Ludwig's angina are emphasized on the basis of data compiled from the literature.

The incompetency of the explanations usually given for the predominant role of the molar teeth in the genesis of Ludwig's angina is shown particularly on account of anatomic studies.

In an attempt to find a solution of the problem of the genesis of this disease, anatomic examinations were undertaken to determine the relation of the roots of the molar teeth to the mylohyoid ridge. These showed that the second and third molar teeth almost invariably reach as far or even below the mylohyoid ridge, that the first molar tooth in most instances reaches a point above the ridge and that the tips of the roots of the anterior teeth are exclusively above this ridge. These findings explain the fact that an infection arising from an infected molar tooth, especially a second or a third one, usually extends into the space below the mylohyoid muscle.

Statistics concerning the involvement of the three molar teeth in the etiology of Ludwig's angina were compiled from the literature. They show a striking similarity to these anatomic findings.

Attention is called to the report of an autopsy in a case of Ludwig's angina which showed a perforation of the inner plate of the mandible below the mylohyoid ridge which resulted from a carious process in a second lower molar tooth. In another case, in which the infection started from a premolar tooth, the perforation was located above the attachment of the mylohyoid muscle. The result here was sublingual cellulitis. These reports are recalled because they represent clinical support for the anatomic findings mentioned.

Dr. Samuel Iglauer made many helpful suggestions owing to his interest in the problem. Drs. Samuel Brown and Archi Fine made the roentgenograms.

11½ East Eighth Street.

Case Reports

PRIMARY CARCINOMA OF THE LEFT SPHENOID CAVITY

GEORGE C. HARDIE, M.B. (TORONTO), JACKSON, MICH., AND CAPTAIN J. H. AHRONHEIM, MEDICAL CORPS, ARMY OF THE UNITED STATES

Malignant growths of the sinuses are not common. Of almost 400 cases of all types and locations of malignant neoplasms collected by one of us (J. H. A.), the sinuses were involved in only 3 cases, in 2 of which the growth originated in the antrum and in 1 in the left sphenoid sinus. The occurrence of malignant tumors of the sphenoid sinus is sufficiently rare to warrant the publication of a case.

Of 63 carcinomas and 7 sarcomas of the sinuses collected by Szende,[2] none originated in the sphenoid sinus. According to Salinger,[3] only 40 cases of malignant growths of the sphenoid sinus had been reported in the literature up to 1939. He pointed out, however, that as a rule carcinoma involving this sinus is diagnosed so late that it is difficult to determine exactly where the neoplasm arose. This suggests that a number of tumors which should have been diagnosed as cancer of the sphenoid sinus were called something else and vice versa. This would be true in the advanced stage of any malignant growth involving the base of the skull. One should, therefore, make no conclusive statements on the basis of the postmortem observations alone but should consider the clinical course and the direction of expansion of the neoplasm before arriving at a final diagnosis.

REPORT OF A CASE

History.—A white woman aged 30 underwent tests for duodenal ulcer in February 1939. During the course of this procedure she complained of "a tooth bothering her." The left upper first molar was removed, but a fistula developed into the left antrum. Roentgenograms of the sinus taken at that time revealed nothing abnormal. Severe pain, localizing about the left ear and the left mastoid region, continued throughout the summer.

A submucous resection was done in September 1939, followed by a radical antrotomy a month later. Histologic examination of material obtained at the second operation revealed no neoplasm. One day after the radical antrotomy paralysis of the left sixth nerve suddenly developed. A caloric test and complete examination of the spinal fluid gave normal results. A blood count showed 7,700 white cells, 4,840,000 red cells and 13.3 per cent hemoglobin. In November 1939 the posterior half of the middle turbinate was removed; the posterior ethmoid cells were exenterated, and a large opening into the left sphenoid sinus was made. This sinus was observed to be filled with pus, and the mucosa was edematous. The results of biopsy of tissue removed from the sinus were negative for neoplasm. Two days later opening of the left mastoid revealed that the cells were intact but were lined with a grayish mucous membrane and contained serous exudate.

After the last operation the patient improved slightly. The pain was not quite so severe, and papilledema, seen prior to the operation, had cleared up. The visual field of the left eye showed a slight temporal defect. There were also some edema of the orifice of the eustachian tube and conductive deafness. During the following months the pain increased. Biopsy of material removed from the sphenoid sinus on two occasions did not reveal neoplasm.

In May 1940 a radical external frontoethmosphenoidectomy was performed. During the course of the operation the anterior ethmoid cells were cleaned out, and the floor of the frontal sinus was removed; these sinuses appeared normal. In a large posterior ethmoid cell the posterior wall was eroded. The anterior wall of the sphenoid sinus was partially gone, as a result of a previous operation, and the lumen was filled with bloody tissue, which was adherent to the bone and pulsated synchronously with the carotid pulse. The bony floor of the sinus was intact. The sinus was cleaned out as much as proximity to and fear of

1. Footnote deleted by author.

2. Szende, B.: Die bösartigen Geschwülste der Nasennebenhöhlen, Monatschr. f. Ohrenh. **72**:925-936, 1938.

3. Salinger, S.: The Paranasal Sinuses, Arch. Otolaryng. **30**:442-479 (Aug.); 633-675 (Sept.) 1939.

exposure of the cavernous sinus allowed. Histologic examination of the tissue removed revealed a medullary squamous cell carcinoma grade 3 with infiltration to, but not into, the bone.

The patient received twenty-two daily roentgen treatments over a period of thirty days with the following dose: 200 kilovolts; 500 r; 50 cm. focus-skin distance; 10 by 10 cm. portal; Thoreus filter; half-value layer 2.55 mm. of copper; effective wavelength 0.092 angstrom units, and 10 r per square millimeter for twenty minutes. The patient's condition improved slightly. The pain decreased, and her left eye rotated laterally about 15 degrees. Vision remained normal. By early fall her symptoms recurred and were more intense than ever before. The pain could be relieved only by use of narcotics. She died on Nov. 19, 1940, twenty months after the onset of the disease.

Autopsy.—External Examination: The body was that of an emaciated white woman, who appeared older than the stated age of 32 years. The weight was approximately 110 pounds (49.9 Kg.). The skin was very pale and showed evidence of recent loss of weight.

Head: The scalp, skull cap and dura mater appeared normal. There was no increase in subdural fluid. The brain weighed 1,250 Gm. and was extremely pale, but showed no gross pathologic condition. Examination of the base of the skull revealed that from the

Fig. 1.—Low power photomicrograph, showing arrangement of neoplastic cells in nests. Anaplasia is indicated by the great number of hyperchromatic nuclei and occasional giant cell forms. Some cell nests have undergone central necrosis, with deposits of cholesterol crystals.

posterior processes to the foramen magnum the bone was soft and largely replaced by soft neoplastic masses. These masses penetrated the dura in the clivus of Blumenbach. Here a neoplastic mass surrounded and embedded the sixth cerebral nerve on the left, where it enters Dorello's canal. The mass extended laterally into a small portion of the anterior aspect of the left petrous bone. It filled the left cavernous and sphenoid sinuses. Both posterior clinoid processes were destroyed. The neoplasm extended close to, but not into, the pituitary body. The right cavernous and sphenoid sinuses were not involved. Posteriorly, the mass extended to the foramen magnum, narrowing its lumen and slightly compressing the medulla oblongata. The left anterior portion of the atlas was completely destroyed. Caudally, the mass reached superficially into the ethmoid bone, a part of which appeared to be destroyed. When the mass was traced farther down, the soft palate became visible but was not involved. Both orbits were free from neoplasm.

Thorax: The heart was hypoplastic, weighing 210 Gm. In the upper lobe of the right lung was an abscess, measuring 2 inches (5 cm.) in diameter and filled with foul pus. Over

this abscess there was an area of localized fibrinous pleuritis. Multiple areas of purulent lobular pneumonia were noted in the lower lobes of both lungs. The organs of the neck appeared essentially normal.

Abdominal and Pelvic Organs: Old pelvic adhesions of peritoneum were present. A small healing duodenal ulcer was noted close to the pylorus. The other abdominal organs appeared essentially normal on gross examination. The pelvic organs were essentially normal.

Microscopic Examination.—Examination of tissue from the sphenoid sinus showed a poorly differentiated squamous cell carcinoma with multiple mitotic figures. There was local infiltration into the left sixth cranial nerve. Fibrosis and active chronic inflammation appeared between the cell nests. Study of the lungs showed patchy acute purulent lobular pneumonia and bronchitis and a large pulmonary abscess with gangrene. Organisms from the mouth and other foreign material were present in the exudate of the pulmonary abscess. A small ulcer, practically healed, was noted in the duodenum. Examination of the pituitary body, heart, aorta, esophagus, thymus, trachea, spleen, large and small intestines, appendix, adrenals, liver and kidneys revealed that they were essentially normal.

Neuropathologic Examination (Dr. K. Scharenberg): Study of the brain revealed no pathologic condition.

Fig. 2.—Sagittal section of the skull, showing the sphenoid sinus. The arrows indicate the five possible routes of extension of malignant growths in the sphenoid sinus.

Diagnosis.—The final diagnosis was poorly differentiated squamous cell carcinoma arising from the mucosa of the left sphenoid sinus and infiltrating into the ethmoid, petrous and occipital bones on the left side; neoplastic involvement of the left abducens nerve; partial compression of the medulla oblongata; partial neoplastic destruction of the atlas; large abscess in the upper lobe of the right lung, due to aspiration of material; patchy acute purulent lobular pneumonia and bronchitis; localized acute fibrinous pleuritis; pulmonary emphysema; marked fatty infiltration of the liver; duodenal ulcer, which was practically healed; old pelvic adhesions, and advanced emaciation and anemia.

COMMENT

In most features this case does not differ greatly from other cases of malignant growths of sphenoid sinus reported in the literature. The early age of the patient is not unusual. Of 13 patients with malignant tumors of the sphenoid sinus whose ages were stated in the report, 3 were in their thirties, while only 1 was 60, the average age being 44. Histologically, the case presented falls under the most common type of squamous cell carcinoma.

In one respect, however, namely, the course of extension which the neoplasm had taken, this case differs from all other cases of similar lesions reported.

In general, it may be stated that five routes of extension, alone or in combination, occur: (1) orbital extension, which is most common and causes exophthalmos; (2) nasal extension, producing obstructive symptoms, epistaxis and, in advanced stages, palpable or even visible masses in the nasopharynx; (3) cranial extension, with involvement and atrophy of the optic nerves and very occasional olfactory implication (extension to the meninges and brain is rare); (4) petrous extension, a route more common than the anatomic relations suggest, resulting in disturbance of the seventh and eighth nerves, and (5) occipital extension, as an example of which the present case seems to be the only one on record.

Considering that in the case of the last route of extension the neoplasm has to grow through the entire body of the sphenoid bone, the infrequency of this route is understandable. With the other paths of extension, with the exception perhaps of the petrous, the neoplasm has to penetrate only thin bony plates before it reaches organs involvement of which produces characteristic symptoms. Logically, combinations of two or more of these five routes of extension are possible and will produce a variety of symptoms.

DIAGNOSIS

The suspicion of a malignant growth in a sinus is usually raised after an extended period of unsuccessful treatment and observation in a case of "chronic sinusitis." Of course. this does not justify the suspicion of cancer in all cases of chronic disease of a sinus. However, when symptoms indicate involvement of neighboring structures, more extensive examination, such as roentgenographic studies or biopsy, become necessary.

In making a diagnosis as to location, one must keep in mind that no conclusive statement can be made from the pathologic observations alone, but that the clinical information is indispensable. In the present case, a neoplasm originating in Rathke's pouch could not have been excluded without the surgeon's information that the floor of the sphenoid sinus was intact at the time of the operation; the floor of the sinus would have been destroyed if the neoplasm had infiltrated from below.

When obtaining material for biopsy, one should not rely on the first specimen alone. As in the case presented, the neoplasm may not be easily accessible, and two, or even more, biopsies may be necessary to establish a correct diagnosis.

HISTOLOGIC TYPES

Histologically, the case presented falls under the most common type of squamous cell carcinomas. However, occasional reports of other histologic types occur in the literature. Handke [4] reported a case of lymphosarcoma in which the picture of rhinogenic meningitis was presented. Autopsy revealed that the neoplasm had infiltrated to the tip of the pyramid of the right petrous bone and around the pituitary body. In Spencer's [5] case the tumor was diagnosed as round cell sarcoma, and it extended forward and laterally. The only report of adenocarcinoma of the sphenoid sinus comes from Calamida,[6] in whose case autopsy revealed infiltration into the sella turcica. Dombrowski [7] reported a case of a papillary type of carcinoma, without further statement as to the exact classification. Behring and

4. Handke, F.: Lymphosarkom der Keilbeinhöhlenschleimhaut unter dem Bilde einer rhinogenen Meningitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **41**:44-51, 1936.

5. Spencer, J. H., and Gall, H.: A Case of Sarcoma of the Sphenoid, J. Roy. Army M. Corps **40**:454-455, 1923.

6. Calamida, U.: Carcinome du sinus sphénoïdale, Arch. internat. de laring. **19**:385-388, 1905.

7. Dombrowski: A Case of Primary Carcinoma of Sphenoidal Sinus, Am. J. Ophth. **12**:225-237, 1895.

Wicherkiewicz,[8] a chlorosarcoma; Schwidop,[9] a spindle cell sarcoma. An unusual histologic type was described by Rossing,[10] who recorded a case of mixed tumor of the salivary gland with destruction of the petrous and sphenoid bones.

It should be borne in mind that the histologic classification in the individual case is partly a matter of personal interpretation, and that poorly differentiated neoplasms might be classified differently by different investigators.

ETIOLOGIC FACTORS

In none of the cases reported in the literature were there any factors which might be considered of etiologic significance in the development of neoplasms of the sphenoid sinus. The only case in which a preceding injury was recorded was that of Handke,[4] but the interval of thirty-six years made any relation between trauma and neoplasm seem highly improbable.

SUMMARY

A case of a poorly differentiated squamous cell carcinoma of the left sphenoid sinus in a woman aged 30 is reported. In a discussion of the possibilities of extension of malignant neoplasms of the sphenoid sinus, it is pointed out that the case presented is the only one on record in which there was infiltration of the neoplastic cells through the body of the sphenoid bone into the foramen magnum.

290 West Michigan Avenue.

8. Behring and Wicherkiewicz, B., cited by Dombrowski.[7]

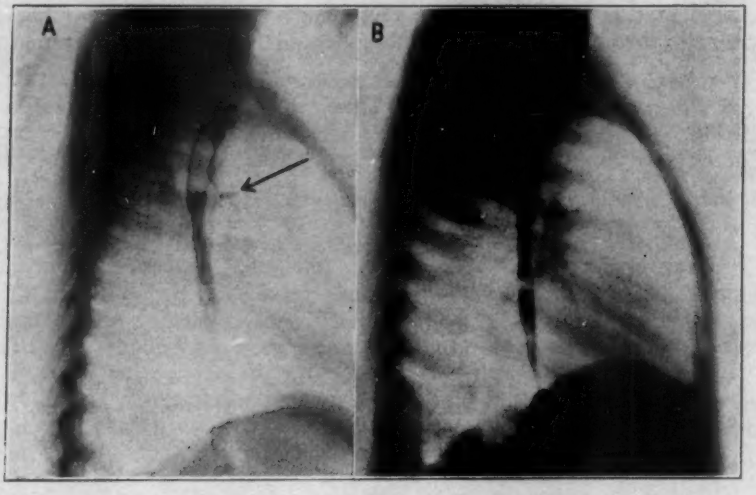
9. Schwidop: Ein Fall von Sarkom der Schädelbasis mit secundärer Affektion des Schläfenbeins, Arch. f. Ohrenh. **35**:39-54, 1893.

10. Rossing, F.: Ein primärer maligner Tumor des Felsenbeines vom Typus der Speicheldrüsenmischgeschwülste, Beitr. z. path. Anat. u. z. allg. Path. **100**: 582-597, 1937.

FOREIGN BODY, JACKSTONE, IN THE ESOPHAGUS: PERFORATION, MEDIASTINITIS, RECOVERY

HARRY L. BAUM, M.D., DENVER

H. McC., aged 2, was brought to Children's Hospital at Denver from Lead, S. D., with a history of having swallowed a jackstone one week previously. Two unsuccessful attempts to remove the foreign body had been made, and as a result the boy was in a serious condition, with high fever and rapid pulse. Physicial examination showed interstitial emphysema of the neck and of the supraclavicular fossae, with some mottling of the skin in the latter regions due to subcutaneous hemorrhage, presumably from a laceration in the esophagus. Roentgenograms showed the presence of a jackstone in the esophagus at the level of the manubrium. Mediastinal emphysema was seen to be present, extending downward around and below the heart and upward into the neck. Roentgenograms taken subsequently, after the patient had ingested a small amount of barium sulfate, showed evidence of an opening in the esophagus, with some of the swallowed barium sulfate in the mediastinum in intimate relation to the trachea (fig.). No barium sulfate entered the trachea from the madiastinum.



A, roentgenogram made before removal of the jackstone and after ingestion of barium sulfate. An arrow indicates leakage into the mediastinum extending from the esophagus to the trachea.

B, roentgenogram made with barium sulfate, three days subsequent to removal of the jackstone. There is no further evidence of leakage. The barium sulfate in the mediastinum has disappeared.

With the patient under ether anesthesia an esophagoscopic examination was made, and it was seen that the esophagus had been badly lacerated in the attempted removals. The perforation could not be identified without more manipulation than was thought justifiable. The esophagus showed much local reaction, with swelling, so that the foreign body was firmly fixed. Therefore, no effort was made to remove it, as it was thought that such manipulation would result fatally. This impression was strengthened by the fact that cough during anesthesia and esophagoscopy visibly increased the interstitial emphysema. This raised the question of possible tracheal perforation also, but an opening was never demonstrated, although one may have been present.

In spite of continued fever, the temperature ranging between 100 and 103 F., and a pulse rate of 130 to 150, the patient did not become highly toxic. His white cell count remained in the vicinity of 10,000 cells, with an average of about 60 per cent polymorphonuclears. He did fairly well clinically, with gradual improvement in his general condition, and it was considered wise not to do anything surgical but to observe him until such time as his

clinical condition justified an attempt at removing the foreign body. The emphysema disappeared in about twelve days, but the ecchymosis lasted for a somewhat longer period. The progress of the mediastinitis was favorable, with steady subsidence of the temperature and of the pulse rate.

The patient's nourishment consisted of sterile liquids, the swallowing of which caused no embarrassment. In fact, there was no evidence of extension of the mediastinitis, although it is assumed that small amounts of liquid must have leaked from the esophagus in the same way that the barium sulfate can be seen to have leaked in the roentgenograms.

At the end of seventeen days the boy's condition was such that it was considered justifiable to do another esophagoscopic exploration with the intention of removing the jackstone if the local inflammatory condition permitted. At that time it was found that the inflammatory reaction had subsided to such an extent that the foreign body was no longer tightly impacted. It was therefore removed. Another roentgenogram was made with barium sulfate three days after the removal of the jackstone (*B* in the figure). It showed no further leakage, and although there was still slight local esophageal irregularity, due to laceration, there was no apparent interference with the swallowing of food, and the further convalescence was uneventful. The patient returned to his home entirely recovered twenty-six days after entering the hospital and thirty-three days after swallowing the jackstone.

COMMENT

The complication of esophageal perforation with mediastinitis and eventual recovery without mediastinotomy is somewhat unusual. The case illustrates the wisdom of leaving a foreign body in place in the esophagus for a time under special circumstances. It further demonstrates that it is sometimes possible to withold mediastinotomy and still achieve a successful result. External drainage extending to the site of perforation would in many similar instances be imperative, but this case shows that such drainage is not necessary in every instance. If this patient's progress had been less favorable, immediate mediastinotomy would have been done. With the evident improvement in his general condition from day to day it was considered justifiable to postpone mediastinotomy, and this very serious procedure was thereby avoided.

510 Republic Building.

Clinical Notes; New Instruments and Technics

A NEW NASAL CLEANSING AGENT

Louis E. Silcox, M.D., Philadelphia

In long-standing cases of chronic sinus disease and atrophic rhinitis with intranasal crust formation and in some cases of postoperative sinus trouble in which there is extensive dryness with crusting, the problem of dealing effectively with this accumulated debris is a major one. The dried secretions and crusts in the nasal cavities and sinuses act as an ideal culture medium for bacteria, and in a short time an offensive odor is present, in many cases amounting to definite ozena.

Ordinary cleansing of the nose with water, a saline solution or some antiseptic solution has been done by the patient as well as by his physician but with unsatisfactory results. The solutions remove the crusts by a mechanical process and in this way remove also any normal mucus present. The removal of crusts is usually incomplete, and as the remaining ones accumulate there is further drying of the underlying mucous membrane with more crust formation, and soon a purulent focus is established with pathologic changes in the underlying membrane.

Various oily preparations have been used for the removal of crusts and accumulated debris with some success, but the quantities used in a complete cleansing are such that lipoid pneumonia or other pulmonary changes are potential hazards.

Because of the physiologic action of the aluminum hydroxide gel and its adsorptive phenomena, it was thought that as a cleansing agent without drying effect on the membranes or any potential pulmonary threat it would offer an advantage over the preparations routinely employed in the care of these disorders.

Twelve patients were treated with aluminum hydroxide gel by placing 1 dropperful of the preparation undiluted in the nose with the head in the Proetz position so that all surfaces of the nasal mucosa would be covered. This was done three times daily, and the results were enlightening. The dry crusts which had become so offensive with ordinary therapy disappeared in from two to three weeks of treatment and the condition of the underlying mucous membrane was greatly improved.

In no case did the aluminum hydroxide gel produce irritation or complications. Chronic sinusitis, which was present in most of these patients, was not improved.

The use of aluminum hydroxide gel or similar colloidal suspensions of aluminum compounds is suggested as an aid in the cleansing of the nasal cavities in chronic sinusitis, atrophic rhinitis or postoperative nasal conditions.

1520 Spruce Street.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

TUMORS OF THE NOSE AND THROAT

GORDON B. NEW, M.D.
AND
EDWARD L. FOSS, M.D.
ROCHESTER, MINN.

In an attempt to evaluate roentgen rays as used in the treatment of certain benign otorhinologic conditions Ersner [1] said that physicians must not allow themselves to become too enthusiastic over any one form of therapy. Radiation is not a panacea and is not effective in all cases, even though some gratifying results have been obtained. Its use is frequently instituted haphazardly or empirically in conditions which are not suitable for such treatment and in which the end results are naturally failures. However, if it is employed rationally and in selected cases, the desired effect may be obtained. He discussed the use of roentgen rays in the treatment of such conditions as cellulitis and eczema of the external auditory canal, deafness of conductive type due to an excess of lymphoid tissue about the eustachean orifice, acute otitis media, acute mastoiditis, sinusitis, adenitis and abscess of the parotid gland.

Some tumors, benign as to cellular type, might be classified as malignant if by their situation they constitute a threat to life or to vital anatomic structures. Motley [2] reported 5 cases in which though the cellular type of the tumor was benign the tumor was situated in a so-called malignant position.

In a comprehensive review of cancers of the pharynx and the larynx, Figi [3] concluded that irradiation is the treatment of choice for cancer of the nasopharynx. As in most instances the lesion is of a high grade of malignancy, the outlook is gloomy. Fibroma of the nasopharynx is benign microscopically but clinically is malignant; electrocoagulation and interstitial irradiation of the growth offer a good prognosis. Most of the cancers of the tonsils and the oropharynx are diagnosed as epithelioma or lymphosarcoma, both highly malignant. A small percentage are found to be low grade epithelioma, mixed tumor or sarcoma of a special type. The more active of the lesions diagnosed as epithelioma should be electrocoagulated and radium points immediately inserted into the coagulated region. The lymph nodes are removed surgically, and this procedure is supplemented by irradiation of the affected surface. In some instances radon seeds are implanted in the involved lymph nodes directly through the intact skin or after surgical exposure of the lymph nodes. The mixed tumor occurring in this situation is encapsulated, grows slowly and does not undergo ulceration. It is best excised through the mouth or by an external

From the Section on Laryngology, Oral and Plastic Surgery, Mayo Clinic.

1. Ersner, M. S.: Roentgen-Ray Therapy for Benign Otorhinologic Conditions, Pennsylvania M. J. **44**:1418-1422 (Aug.) 1941.

2. Motley, F. E.: Position Malignancy, Arch. Otolaryng. **34**:771-786 (Oct.) 1941.

3. Figi, F. A.: Cancer of the Pharynx and Larynx, S. Clin. North America **21**:1027-1035 (Aug.) 1941.

excision. A malignant tumor of the base of the tongue and hypopharynx is usually found to be highly malignant squamous cell epithelioma, lymphosarcoma, or adenocarcinoma. If it is encountered early enough, electrocoagulation may be justified but in most cases interstitial irradiation is the treatment of choice. The treatment of cancer of the larynx depends on a number of factors. The extent of the growth as well as its activity must be considered. Previous treatment and acute inflammatory reaction in the tumor are also of importance, as both render wide removal imperative. Operation is the treatment of choice in cases in which carcinoma is limited to the interior of the larynx. Removal by suspension laryngoscopy should not be attempted unless the growth is readily and completely visible by the method. Thyrotomy and laryngectomy are the procedures commonly employed in the removal of a malignant tumor of the larynx.

Cutler [4] has devised a new technic for the use of radiation from an external source which he said has a more pronounced effect on the more radioresistant cancers of the mouth, pharynx and larynx than do the methods now in use. This technic has been called the method of concentration. Since this method has been used only three and a half years, the author made no statements as to the permanence of the results. In a later article Cutler [5] discussed radiosensitivity of tumors and considered those which fall into the radiosensitive group and those which fall into the radioresistant group. He stated, however, that no sharp line can be drawn between radiosensitive and radioresistant tumors.

TUMORS OF THE NOSE AND ACCESSORY SINUSES

In a general consideration of dermoid cysts, Parkinson [6] stated that the dermoid of the nasal bridge arises, as do dermoids situated elsewhere, from epidermal cells included or sequestrated during embryonic development. By the time the growth is clinically recognizable, there is commonly close association between the wall of the cyst and the nasal mucosa, owing to contiguity. Although instances of direct connection between the dermoid wall and the dura are not recorded in the literature, instances of close association owing to extension toward the frontal lobe are mentioned. Secondary infection sometimes occurs, probably from trauma. Thus a certain hazard is associated with removal, and various suggestions have been made for the development of an effective yet safe surgical technic. He reported a case of dermoid cyst of the bridge of the nose in a girl 4 years of age. This tumor was successfully removed by sharp dissection in one operation.

Rosedale [7] reported a case of dermoid cysts of the bridge of the nose. The patient was a child 4 years of age. This tumor was removed surgically. He also considered the gross pathologic aspects, the microscopic anatomy, the incidence, the treatment, the diagnosis and the theory of development of these cysts.

Solitary polyp of a nasal choana occurs relatively infrequently; usually the solitary polyp arises in the maxillary sinus, is commonly of the fibromyxomatous type and has a smooth surface. Pastore and Lillie [8] reported a case of osteofibro-

4. Cutler, M.: Concentration Method of Radiotherapy for Cancer of the Mouth, Pharynx and Larynx, J. A. M. A. **117**:1607-1610 (Nov. 8) 1941.

5. Cutler, M.: Concentration Method of Radiotherapy for Cancer of the Mouth, Pharynx and Larynx, Cincinnati J. Med. **23**:117-124 (May) 1942.

6. Parkinson, S. N.: Dermoid Cyst of the Nasal Bridge, Arch. Otolaryng. **34**:829-831 (Oct.) 1941.

7. Rosedale, R. S.: Congenital Dermoid Cyst of the Nose, Ohio State M. J. **38**:132-134 (Feb.) 1942.

8. Pastore, P. N., and Lillie, H. I.: Unusual Polypoid Tumor of the Nasal Choana, Proc. Staff Meet., Mayo Clin. **16**:730-732 (Nov. 12) 1941.

adenoma. The patient was a man aged 59. This tumor was polypoid in type and was removed through the oral approach with a snare loop.

A nasal tumor composed wholly or in part of brain tissue is rare but may occur. Schwartz and Isaacs [9] said that it may appear in any part of the nose. It is always congenital. It usually shows little or no tendency to grow, and there is no record of metastasis in any instance. It may be intranasal or extranasal and may or may not have a direct connection with the brain. A great number of terms have been used in describing nasal neoplasms of this type. The authors reported a case of astrocytoma. The patient was a boy 13 months of age. The mass was removed with a snare and punch forceps, and the base of the tumor was curetted thoroughly.

Salinger [10] reported a case of cavernous hemangioma of the nose, nasal septum and forehead. The patient was a woman 20 years of age. The size of the masses was reduced by preliminary interstitial radiation. Subsequently portions of tumor masses were removed surgically.

In an excellent review of carcinoma of the nose, nasopharynx and paranasal sinuses, Havens [11] said that carcinoma involving the nose, nasopharynx and paranasal sinuses frequently is well advanced before discovery, because the lesion may remain symptomless for a considerable time and because the first symptoms, particularly of a lesion of the nasopharynx, may be referred to some other structure, such as the neck, the eye or the ear. Carcinoma of the nose is most likely to be found on the septum or high on the lateral wall of the nasal passage. Recent nasal obstruction, increased nasal discharge or purulent discharge with bleeding should bring the possibility of malignant disease to mind. A carcinomatous mass found in the nasal passage may be primary there or it may have extended into the nose from the maxillary or the ethmoid sinus. The author stressed the fact that biopsy should be performed by the physician who is to treat the malignant lesion, if it is present. The choice of treatment for a cancer within the nasal passages will depend on the site and the extent of the lesion as well as on the microscopic classification. Suggestions were made for the criteria to be used in the choice of proper treatment. Symptoms of carcinoma of the paranasal sinuses may be nasal obstruction, obstruction of the lacrimal duct, increased unilateral nasal discharge, loosening of upper teeth in the cuspid to molar regions or bulging of the cheek. If the usual methods of examination, including the roentgenographic, do not remove all suspicion of malignant disease, an exploratory operation should be performed. Treatment with surgical diathermy and radiation was outlined. This affords the patient a 35 to 40 per cent chance of cure. Symptoms of carcinoma of the nasopharynx may be enlargement of a lymph node in the upper part of the midcervical region, deep-seated pain in the temporoparietal region, ringing or roaring in the ears, loss of hearing in one ear or double vision. Other less common symptoms are considered. The type of carcinoma found most frequently in the nasopharynx is squamous cell epithelioma, usually grade 4. Lesions of this type are radiosensitive. and irradiation is the treatment of choice. In order to be successful, the management of carcinoma of the nose, paranasal sinuses and nasopharynx demands seasoned diagnostic and therapeutic judgment. Since the lesion usually cannot be visualized

9. Schwartz, A. A., and Isaacs, H. J.: Glioma (Astrocytoma) of the Nares, Arch. Otolaryng. **34**:838-843 (Oct.) 1941.

10. Salinger, S.: Cavernous Hemangioma of Nose, Nasal Septum and Forehead, Ann. Otol., Rhin. & Laryng. **51**:268-272 (March) 1942.

11. Havens, F. Z.: Carcinoma of the Nose, Nasopharynx and Paranasal Sinuses, S. Clin. North America **21**:1009-1015 (Aug.) 1941.

readily, physicians in general must be alert in order to recognize early the symptoms and signs suggestive of malignant disease involving these structures.

Watson [12] reviewed 127 cases of cancer of the paranasal sinuses. He outlined three plans of treatment. Plan A is based on the principle of quick irradiation by interstitial implantation of gold radon seeds, followed by wide cautery excision of the tumor and its condemned surrounding bony structures. Plan B also is a combination of irradiation and operative treatment. In this plan, however, the major responsibility falls on external radiation, which is usually administered by the divided dose method. Surgical procedures in this plan are conservative and are utilized only when necessary for the control of hemorrhage or for the establishment of drainage for empyema of the accessory nasal sinuses. Plan C is reserved for cases of hopelessly advanced cancer in which prolonged sepsis and pain have produced a serious state of general debility. A considerable palliative effect can be obtained if the patients are hospitalized, if small doses of roentgen rays are employed and if drainage is instituted conservatively.

Atkins [13] expressed the opinion that a combination of operation and radiation therapy gives much better end results when used in the treatment of carcinoma of the paranasal sinuses than does the use of either surgical procedure or radiation alone. He said that there must be close cooperation between the surgeon and the radiologist.

TUMORS OF THE LIPS AND MOUTH

The several theories as to the origin of the various cysts included under the term "ranula" have been reviewed by Connelly.[14] He said that there is no satisfactory classification at present. Excision is the preferred treatment for all of these cysts. When this is impossible, the Partsch operation is preferable to the more simple measures which can be offered. The author reviewed a case of ranula. The patient was a girl 19 years of age. The cyst was removed by the Partsch operation as follows: The overlying mucous membrane was incised and separated from the cyst. The submaxillary duct was dissected upward. The upper portion or dome of the wall of the cyst was sutured to the cut edges of the mucous membranes. Light gauze packing was placed in the cavity.

Coates [15] reported a case of a schwannoma of the mouth. The patient was a woman aged 44. The tumor was situated under the right side of the tongue. Its presence had been known for four weeks. No difficulty was encountered in removing the growth surgically through the mouth. There has been no recurrence in two and a half years. The term "schwannoma" is used in the article because the pathologist who made the diagnosis reported that the growth was schwannoma. Histologically, a tumor of this type is considered to be identical with neurofibroma or neurinoma and may be found on any peripheral nerve anywhere in the body. Such lesions are rare in the mouth or the pharynx.

The literature on mixed tumor of the lip was reviewed by Warshaw.[16] He reported a case of mixed tumor of the upper lip. The patient was a woman 35 years of age. The tumor was removed surgically.

---

12. Watson, W. L.: Cancer of the Paranasal Sinuses, Laryngoscope **52**:22-42 (Jan.) 1942.
13. Atkins, W. L.: Radiation and Surgical Treatment of Malignancy in Paranasal Sinuses, New Orleans M. & S. J. **94**:19-24 (July) 1941.
14. Connelly, E. J.: Ranula, J. Iowa M. Soc. **32**:127-129 (March) 1942.
15. Coates, G. M.: Schwannoma of the Mouth, Arch. Otolaryng. **34**:1166-1167 (Dec.) 1941.
16. Warshaw, D.: Mixed Tumors of the Lip: Report of a Case, M. Rec. **154**:87-90 (Aug. 6) 1941.

Martin and Graves [17] reported 2 cases of bulky plexiform neurofibroma involving the oral cavity. The patients were children, 3 and 5 years of age respectively. The growths were part of a generalized neurofibromatous disease.

Federspiel [18] said that technic for the reconstruction of a lip cannot be standardized and that special study and planning are required in each case. He reported a case of plastic repair for reconstruction of the lower lip. The patient was a man 40 years of age. In this case the lower lip had been entirely removed for the eradication of a tumor diagnosed as epithelioma.

Erich [19] considered in detail the malignant tumors of the mouth, their diagnosis and their treatment. He said that unfavorable results cannot be avoided in some cases but usually are due to one or more of the following factors: (1) failure to recognize the nature of the growth, with a resulting loss of time; (2) application of caustic drugs; (3) insufficient treatment, and (4) neglect of the regional lymphatics. Malignant tumors of the cheeks for the most part are squamous cell epithelioma, and in most instances are of low grade (1 or 2 of Broders' classification). Because of the rich supply of lymph vessels about the buccal mucosa, cervical metastasis can occur early in the course of the disease. These tumors are usually not radiosensitive, and complete destruction may be accomplished with greater assurance by surgical treatment (usually surgical diathermy) than by irradiation of the growth. Consequently, unless the local lesion is very small and of a very low grade of malignancy, a block dissection of the cervical lymph nodes is indicated. In cases of grade 4 epithelioma, however, external irradiation of the neck may be preferable to surgical measures. Since in most cases epithelioma of the jaw is a rather inactive lesion, it is believed to be best destroyed by means of surgical diathermy. Electrocoagulation offers a simple method of treating the involved portion of bone. If the lesion of the jaw is highly active, it will require radiation therapy in addition to surgical procedures. The majority of cancers primary in the upper jaw are of low grade, whereas those developing in the maxillary sinus are inclined to be highly active. In treating the low grade lesions, surgical diathermy is employed. High grade malignant tumors of the maxillary sinus require radiation therapy. Regardless of the form of treatment used, about 10 per cent of patients who have a malignant tumor of the maxillary sinus lose the eye on the involved side. Adenocarcinoma of the mixed tumor type usually occurs in the palate. When it is well encapsulated, excision is an effective method of removal. However, should the capsule not be intact, it is well to destroy the tumor by thorough electrocoagulation. Adamantinoma is a neoplasm of very low grade of malignancy. Although it exhibits an extreme degree of slowness in growth, it recurs promptly when it is removed incompletely and in occasional cases metastasizes to the regional lymph nodes. Satisfactory results may be expected only when very radical surgical measures are undertaken. In general, malignant tumors of the tongue and floor of the mouth are the most serious neoplasms which occur in the oral cavity. Malignant tumor of the tongue is usually more active than those affecting other regions of the mouth and is prone to produce very early cervical metastasis. Most malignant tumors of the tongue and floor of the mouth are diagnosed as squamous cell epithelioma.

17. Martin, H. E., and Graves, C. L.: Plexiform Neurofibroma (von Recklinghausen's Disease) Invading the Oral Cavity, J. A. M. A. **117**:1535-1539 (Nov. 1) 1941.

18. Federspiel, M. N.: Extensive Plastic Repair for Restoration of the Lower Lip: Report of Procedure in a Case in Which the Lower Lip Was Entirely Removed for Eradication of Epithelioma, Am. J. Orthodontics (Oral Surg. Sect.) **28**:163-166 (March) 1942.

19. Erich, J. B.: Malignant Tumors of the Mouth, S. Clin. North America **21**:1017-1025 (Aug.) 1941.

The size, the situation and the grade of the tumor determine the type of treatment to be employed in each case of carcinoma of the tongue or of the floor of the mouth. In practically all cases of carcinoma of the tongue, block dissection of the cervical lymph nodes is indicated whether or not these nodes are palpably enlarged.

Three of four chronic ulcers of the mouth are cancerous. This figure was determined by Keyes [20] after a review of 2,077 case histories. Of these, 1,561 were histories of malignant lesions, an incidence of 75.2 per cent. Of this series of malignant tumors, 28 were diagnosed as sarcoma, 8 as adenocarcinoma, 2 as basal cell carcinoma and the remaining 1,523 as squamous cell carcinoma. A complete review of the diagnostic features of the 1,523 cases of ulcer diagnosed as squamous cell carcinoma of the mouth was given. Cancer may spread from such an oral lesion to the submaxillary, buccal or anterior parotid group of lymph nodes, sometimes years after cure of the primary tumor. This spread may be homolateral, contralateral or bilateral. From the buccal mucosa cancer spreads first usually to these groups of nodes, always homolaterally, at a rate generally faster than from the lip. From the tongue carcinoma usually spreads to the jugulodigastric lymph nodes and almost as quickly to the remaining nodes of the deep jugular chain. Extension generally is homolateral but often is contralateral or bilateral, and at a more rapid rate than cancers of other sites in the mouth. Biopsy was often used to confirm the diagnosis of cancer of the mouth. Occasionally a lymph node in the neck was removed. The author called attention to the fact that biopsy itself is not always perfect. There is a definite chance for error in cases in which the patient has previously been treated with radium. The most difficult differential diagnosis is between radiation ulcer of the mouth and cancer.

Because of the abundant literature arguing against the value of the excision of lymph nodes in cases of intraoral cancer, Kennedy [21] has written an excellent paper in which he brought out the fact that prophylactic treatment of lymph nodes in the neck is frequently done in such a half-hearted manner that it is useless. Except in radiosensitive, grade 3 and 4 lesions, there is little evidence that external irradiation has a permanent arresting effect on squamous cell carcinoma which has metastasized to the cervical lymph nodes. Removal of the lymph nodes of the neck should not be undertaken in cases of grade 3 or 4 carcinoma or in cases of epithelioma. The fact that cancer cells are not found in nodes removed does not rule out the presence of metastasis unless serial sections of all nodes have been made. Metastasis may be present even though no lymph nodes are palpable. Palpable lymph nodes do not necessarily prove the presence of metastasis. The direction of metastatic spread of cancer of the mouth is more definite and localized than that of cancers of other sites; therefore, the plan of approach to excision of lymph nodes needs to be varied with the site of the lesion about the mouth. Radiation therapy has apparently improved but little the results in cases of metastatic involvement of the cervical lymph nodes. The incidence of five year arrests of intraoral cancer will show little improvement with present known methods of care until the defeatist attitude toward treatment of metastatic involvement of the lymph nodes has been overcome.

Martin, MacComb and Blady [22] have made an analysis of 375 consecutive cases of cancer of the lip. In all of these cases the diagnosis was proved histo-

20. Keyes, E. L.: Chronic Ulcerative Lesions of the Mouth: Incidence of Cancer in 2,077 Cases at Barnard Hospital, Am. J. Surg. **56**:70-78 (April) 1942.

21. Kennedy, R. H.: Management of Lymph Nodes in Neck, Metastatic from Carcinoma of the Mouth, Ann. Surg. **114**:813-819 (Nov.) 1941.

22. Martin, H. E.; MacComb, W. S., and Blady, J. V.: Cancer of the Lip, Ann. Surg. **114**:226-242 (Aug.); 341-368 (Sept.) 1941.

logically. All cases of cancer of the lip observed from 1928 to 1934 at the authors' clinic were included. They discussed the causes, the pathologic aspects, the symptoms, the treatment and the prognosis of cancer of the lip. The net percentage of five year cures in these 375 cases was 70. In about half of the cases the lesion was grade 2 squamous carcinoma and in a third of the cases it was grade 1, with grades 3 and 4 making up less than 2 per cent. As a rule, in cases of cancer of the lip metastasis occurs later in the course of the disease than it does in cases of cancer of the tongue, of the floor of the mouth or of the tonsil. In about 37 per cent of the cases metastasis occurred sometime during the course of the disease. Except when the lesion arises on the upper lip, the position of the first lymph node involved and the later progress of cervical metastasis tend to be rather orderly; that is, the nodes most often involved are the submaxillary nodes (more than 50 per cent of cases in which metastasis occurs) or the submental nodes (about 8 per cent) of the corresponding side, with orderly progression from these nodes to the upper deep cervical nodes and later to the middle and lower deep cervical nodes. Either irradiation or operation can be employed with equally good chances of cure in the uncomplicated primary lesion. The selection of the method of treatment in the individual case must therefore be made on some basis other than the supposed superiority of one or the other method from the standpoint of cure alone; the most important considerations are the cosmetic result, the expediency of one particular method as regards its availability, the training and the experience of the surgeon, the period of morbidity and the general condition of the patient. The authors did not approve of prophylactic dissection of the neck and did not feel that the application of small prophylactic doses of roentgen rays to the neck is of any use or that large doses are justified.

A series of 318 cases of cancer of the lower lip were reviewed by Lamb and Eastland.[23] The patients were treated by roentgen rays and radium. The five year survival rate in these cases was 72.2 per cent. Protracted radium therapy with the element well filtered has produced a higher percentage of cures than did the older technic in which smaller doses were employed at short intervals.

In 74 cases of proved carcinoma of the lower lip observed at the Brooklyn Cancer Institute, a follow-up study was made from six months to six and a half years after therapy had been discontinued. In 59 of these cases the result was listed as successful, and in 15 cases it was listed as a failure. After a survey of these cases, Howes and LaRosa [24] said that operative removal or the application of radium or of roentgen radiation to the local lesion may be equally efficient in producing a cure.

Howes and Bernstein [25] reviewed a group of 138 cases of cancer of the mouth. This series included all cases of oral carcinoma observed at the Brooklyn Cancer Institute since October 1936. Twenty-seven cases have been omitted from the analyses because of insufficient data. The authors said that the cure of this type of cancer is entirely a radiologic problem except for the treatment of complications. The poor end results in cases of intraoral carcinoma were ascribed to (1) the anatomic structure of the oral cavity and associated complications, as osteomyelitis, infection, hemorrhage and early metastasis, (2) the age and the type

23. Lamb, J. H., and Eastland, W. E.: Cancer of the Lower Lip: Treatment with Radiation, J. A. M. A. **117**:600-607 (Aug. 23) 1941.

24. Howes, W. E., and LaRosa, F. J.: Carcinoma of the Lower Lip: Interval Statistical Survey of the End-Results in All Cases Treated at the Brooklyn Cancer Institute, 1930 to 1939 Inclusive, Am. J. Roentgenol. **47**:39-49 (Jan.) 1942.

25. Howes, W. E., and Bernstein, L.: Oral Carcinoma, Radiology **37**:468-475 (Oct.) 1941.

of patients and (3) the large percentage of advanced lesions. They concluded that further intensive study is needed to improve radiologic methods of treatment.

A case of carcinoma of the mucous membrane of the cheek in an infant 4 months of age was reported by Carey.[26] The tumor was surrounded by mucoid material which apparently had escaped from an injured buccal gland in the vicinity. The author expressed the opinion that the mucus may have been a potent factor in the genesis of the tumor.

The general plan of treatment of carcinoma of the mouth was discussed by Wigby.[27] Six cases of intraoral carcinoma were reported. He recommended that any lesion that remains in the mouth for more than two weeks should be investigated by biopsy.

TUMORS OF THE NASOPHARYNX

Schuster [28] reviewed the treatment used in 2 cases of nasopharyngeal fibroma. One tumor was treated by interstitial radiation only and the other by a combination of actual cautery and interstitial radiation. Postnasal fibroma of the juvenile type is a rare tumor of unknown cause. The treatment should control hemorrhage and restore function but should not destroy surrounding tissue. Radiation will often control this tumor, but if the tumor is large and has invaded surrounding structures, operation, electrocoagulation or galvanocautery often is required. Surgical removal is more easily accomplished after radiation has decreased the vascularity of the tumor.

Hill [29] reported a case of fibroma of the nasopharynx in a boy 13 years of age. When the patient first was seen, there was marked intranasal extension of the growth with involvement of the ethmoid sinuses, the orbit and the sphenoid bone. In this case erosion through the roof of the ethmoid sinus, possibly in the cribriform plate, allowed a sudden development of pneumatocele, which disappeared after the patient had been kept in bed for nine days. Irradiation of the tumor with radon implants caused a marked decrease in its size.

Hughes [30] reviewed the literature on fibroma of the nasopharynx and concluded that radium therapy seems to be the treatment of choice. He reported a case of nasopharyngeal fibroma in a youth 19 years of age. The tumor was removed in two stages by the use of a snare.

Titrud and Peyton [31] reviewed approximately 195 cases of nasopharyngeal tumor. They said that the early development of neurologic signs and symptoms should lead to an early diagnosis and the institution of proper treatment. Inasmuch as these tumors are extremely radiosensitive, irradiation is the treatment of choice. In 131 of this series of cases the diagnosis was squamous cell carcinoma, in 37 lymphoepithelioma and in 10 lymphosarcoma; 63 per cent of the patients with squamous cell carcinoma had neurologic symptoms as compared with 73 per cent of the patients with lymphoepithelioma.

---

26. Carey, R. M.: Epidermoid Carcinoma in the First Decade of Life: Report of a Case, J. Pediat. **20**:496-498 (April) 1942.

27. Wigby, P. E.: Intra-Oral Radiation Therapy, Texas State J. Med. **37**:422-428 (Oct.) 1941.

28. Schuster, F. P., and Schuster, S. A.: Postnasal Fibroma, Texas State J. Med. **37**:782-784 (April) 1942.

29. Hill, F. T.: Nasopharyngeal Fibroma, with a Report of a Case with Secondary Pneumocephalus, Ann. Otol., Rhin. & Laryng. **51**:105-111 (March) 1942.

30. Hughes, T. E.: Nasopharyngeal Fibroma: Report of a Case, Arch. Otolaryng. **34**:57-68 (July) 1941.

31. Titrud, L. A., and Peyton, W. T.: Neurological Complications of Nasopharyngeal Tumors, Bull. Minnesota M. Foundation **3**:14 (Nov.) 1941.

Figi [32] reported a case of grade 4 squamous cell epithelioma of the nasopharynx. The patient was a boy 11 years of age. When he was first seen, extensive bilateral metastasis was present in the cervical region. Radium was applied both to the neck and to the nasopharynx. Ten years later the patient was in excellent health.

TUMORS OF THE OROPHARYNX AND HYPOPHARYNX

Hall and Owens [33] reported a case of a solitary neurofibroma of the pharynx. The patient was a man 57 years of age. The tumor was removed through the mouth after ligation of the external carotid artery. The case is reported because of the extreme rarity of this tumor.

TUMORS OF THE LARYNX

Chondroma of the larynx must be distinguished from cancer. If one follows out the usual diagnostic procedure for arriving at the diagnosis of a malignant lesion, a positive diagnosis can usually be made. Orton [34] said that this type of growth may arise from any of the cartilages of the larynx. He reviewed 2 cases. The first patient was a woman 40 years of age who had epidermoid carcinoma of the larynx, confused with chondroma of the larynx. The second patient was a man 66 years of age who had chondroma of the larynx, the first symptoms of which had appeared twenty years previously. For the complete removal of this growth, laryngectomy was necessary.

In a careful study of a series of cases of polyps of the vocal cords, Friedberg and Segall [35] found that the dominant microscopic features of such polyps were congestion, edema, thrombosis and hyalinization. Polyps of the vocal cords develop in patients whose cords have been abused or misused. They are usually seen at the junction of the anterior and middle thirds of the vocal cord. Acute polyps may suddenly develop after severe vocal strain. In some cases they are due to hemorrhage in Reinke's space. Preexisting venous dilatation may be a predisposing factor.

Van Loon and Diamond [36] reported a case of neurofibroma of the larynx. The patient was a girl 17 years of age. Laryngofissure was performed and the tumor mass shelled out by blunt dissection.

An anterior commissure tendon which never had been described previously was found by Broyles.[37] He said that the presence of an anterior tendon and its extension into the thyroid cartilage may explain many of the recurrences of carcinoma after laryngofissure. For this reason he advocated the removal of the anterior ridge or crista of the thyroid cartilage and the anterior portion of the healthy cord along with the diseased portion of the vocal cord.

---

32. Figi, F. A.: Highly Malignant Carcinoma of the Nasopharynx with Bilateral Cervical Metastasis in Child Well After Ten Years, Ann. Otol., Rhin. & Laryng. **50**:1218-1220 (Dec.) 1941.

33. Hall, C., and Owens, H.: Solitary Neurofibroma of the Pharynx, Arch. Otolaryng. **34**:1163-1165 (Dec.) 1941.

34. Orton, H. B.: Chondroma of the Larynx: Report of a Case, Ann. Otol., Rhin. & Laryng. **50**:715-722 (Sept.) 1941.

35. Friedberg, S. A., and Segall, W. H.: The Pathologic Anatomy of Polyps of the Larynx, Ann. Otol., Rhin. & Laryng. **50**:783-789 (Sept.) 1941.

36. Van Loon, E. L., and Diamond, S.: Neurofibroma of the Larynx, Ann. Otol., Rhin. & Laryng. **51**:122-126 (March) 1942.

37. Broyles, E. N.: Anterior Commissure Tendon of the Larynx: Its Significance in the Laryngofissure Operation; Preliminary Note, Bull. Johns Hopkins Hosp. **70**:90-91 (Jan.) 1942.

Clerf [38] said that biopsy is indicated and should be performed promptly if the diagnosis is doubtful after examination of the larynx. Successful treatment of cancer of the larynx is dependent on early diagnosis and prompt extirpation of the growth.

LeJeune [39] reported an interesting case in which a man had undergone ninety-seven operations under general anesthesia. The first operation was performed for multiple papilloma of the larynx when the patient was 2½ years of age. Aspiration pneumonia developed after the ninety-seventh operation, which was performed when the patient was 21 years of age. This operation was performed for squamous cell carcinoma, grade 2.

Multiple myeloma is a relatively rare disease which occasionally invades regions that are treated by otolaryngologists. It manifests itself as a solitary extramedullary tumor or as a part of a general myelomatosis. Metastasis is rare. Gilmore [40] reported a case of multiple myeloma in which metastasis occurred in the larynx.

Salinger [41] reported a case in which irradiation of the larynx in an attempt to treat multiple papilloma was responsible for an arrest of development of the larynx.

It is possible that in the failure to remove a benign lesion one is missing an opportunity to prevent cancer. A benign tumor of the larynx involving the vocal cords should be removed and an attempt made to restore the normal function of the cords. The treatment of cancer of the larynx is also fairly well standardized. The majority of laryngologists believe that laryngofissure should be done in cases of early cancer of the vocal cords and that laryngectomy is the treatment of choice for more advanced lesions involving the vocal cords and adjacent parts. Radiation has been found valuable for extensive lesions that have been operated on and for recurrent growths. It is particularly effective when the recurrence is local and also for cancer in patients past 65 years of age. For most extrinsic cancers in which metastasis occurs early, irradiation is the best treatment. Tucker [42] arrived at the foregoing conclusions after reviewing a series of 1,120 cases of laryngeal tumor. In 670 of these cases the tumor was benign, and in 450 of the cases it was malignant.

In a comprehensive review of laryngeal cancer McCall and Lamb [43] said that in about 90 per cent of cases of cancer of the vocal cords the lesion is of the squamous cell type and does not metastasize early. Syphilis and tuberculosis may occur coincidentally with cancer, and one should perform a biopsy immediately rather than use a therapeutic test. If a diagnosis of carcinoma of the larynx is made early enough and if the growth does not involve the anterior commissure, one may perform laryngofissure and preserve the normal air passage. However, if the growth has become deeply embedded in the tissue or involves the anterior commissure, according to these authors, total laryngectomy offers a better chance

38. Clerf, L. H.: Hoarseness and Cancer of the Larynx, West. J. Surg. **49**:599-602 (Nov.) 1941.

39. LeJeune, F. E.: Story of Warren Bill (97 Operations for Multiple Laryngeal Papillomata and Their Complications), Ann. Otol., Rhin. & Laryng. **50**:905-908 (Sept.) 1941.

40. Gilmore, G. B.: Multiple Myeloma with Laryngeal Metastasis, Arch. Otolaryng. **34**:453-460 (Sept.) 1941.

41. Salinger, S.: Arrested Development of the Larynx Following Irradiation for Recurring Papillomas, Ann. Otol., Rhin. & Laryng. **51**:273-277 (March) 1942.

42. Tucker, G.: Observation of the Inter-Relationship, Diagnosis and Treatment of Benign and Malignant Tumors of the Larynx (with Special Reference to the Prevention and Cure of Laryngeal Cancer), Tr. Am. Laryng., Rhin. & Otol. Soc. **47**:29-34, 1941.

43. McCall, J. W., and Lamb, A. J.: Laryngeal Carcinoma, Ohio State M. J. **37**:1053-1055 (Nov.) 1941.

of permanent cure for the patient. The authors reviewed 16 cases of carcinoma of the larynx in which operation had been performed.

Schall [44] advocated that operation for cancer of the larynx should be performed only after certain rigid requirements have been fulfilled. If the growth is intrinsic and of a low grade of malignancy and if there is no involvement of the lymph nodes, a cure for three to five years will follow total laryngectomy in approximately 75 to 79 per cent of cases.

In an excellent review of the various treatments indicated in cases of laryngeal cancer Wright [45] said that laryngofissure is the ideal operation in cases of comparative early cancer of the vocal cords in which there is no impairment of the motility of the cords and little subglottic or posterior extension. Laryngectomy is indicated for advanced intrinsic lesions without metastasis, and radiation therapy is indicated for all primary extrinsic lesions and inoperable lesions. Direct laryngoscopy should be performed in all cases in which a malignant lesion is suspected and in cases in which a tentative or a definite diagnosis of a cancer has been made by mirror laryngoscopy. It is only by this means that the character and extent of the lesion can be carefully studied and a representative specimen for biopsy accurately and skilfully obtained. Wright [45] reported 18 cases in which operation was performed. Laryngofissure was employed in 7 cases, endoscopic cordectomy in 1 case and laryngectomy in 10 cases. There was no operative mortality. Recurrence occurred in 4 of the cases in which laryngectomy was performed, or in 22 per cent of the entire series of cases reported. There were no recurrences in cases in which laryngofissure was performed.

Quick [46] said that careful roentgen therapy offers an excellent chance for cure of laryngeal cancer uncomplicated by previous treatment. Roentgen therapy is applicable to a larger group of such lesions than is radical operation, is free from the interim risk and results in complete restoration of function. There is, however, a limit beyond which it is unsafe to increase the dose of roentgen rays. Laryngofissure plus use of radon implants or cautery offers a chance for further cure of cancers which have proved to be resistant to radiation or which have recurred. Metastatic involvement of the cervical lymph nodes is treated by surgical exposure and radon implantation; its occurrence is infrequent.

A detailed technic of laryngectomy was described by Gill [47] in a comprehensive article on malignant lesions of the larynx. The diagnosis of cancer of the larynx is based on a complete history, a physical examination, an examination with a laryngeal mirror and, in some instances, direct laryngoscopy. Before operation is advised, biopsy should be done and histologic studies made by a competent pathologist. The author used the Broders classification for the grading of malignancy. A satisfactory biopsy is not an easy operation. Negative reports are sometimes given in cases of advanced cancer. This is due to failure to obtain an adequate specimen or a specimen representative of the whole growth. In cases of subglottic growth it is difficult to obtain a satisfactory view with direct laryngoscopy, and it is necessary to use a short bronchoscope.

---

44. Schall, L. A.: Cancer of the Larynx: Surgical Treatment, Am. J. Roentgenol. **46**:30-32 (July) 1941.

45. Wright, E. S.: Diagnosis and Treatment of Cancer of the Larynx, Ann. Otol., Rhin. & Laryng. **51**:228-249 (March) 1942.

46. Quick, D.: Treatment of Laryngeal Cancer by Irradiation, Am. J. Roentgenol. **46**:11-20 (July) 1941.

47. Gill, E. G.: Cancer of the Larynx, Virginia M. Monthly **68**:509-512 (Sept.) 1941.

Fletcher [48] discussed laryngeal tumors in general. He said that the sheet anchors of the successful treatment of cancer of the larynx are thyrotomy and complete laryngectomy. There should be no delay in cases in which the lesion is considered operative. The grade of malignancy also exercises a profound effect on the prognosis. Some authors have reported good results in cases of intrinsic carcinoma in which operation has been declined, but neither radium therapy nor roentgen therapy hold out good prospects of a lasting cure.

Lenz [49] studied 166 cases of carcinoma of the larynx. He said that the failure to cure cancer of the larynx by roentgen therapy may be due to the following causes: (1) local causes (extensiveness of the growth, invasion of cartilage and deep local infection); (2) general causes, which are associated with the inability of the patient to tolerate treatment, and (3) technical causes, which are due to faulty technic in administering the roentgen rays.

Figi [50] said that the method of treatment advisable in a case of carcinoma of the larynx often cannot be determined until the growth has been inspected directly through a laryngofissure. Thyrotomy is effective in cases in which operation discloses that the malignant lesion is more extensive than it was believed to be. Electrocoagulation is a valuable adjunct in the removal of carcinoma of the larynx by laryngofissure, pharyngotomy or suspension laryngoscopy. Lesions which involve the supraglottic portion of the larynx often can be removed by means of suspension laryngoscopy and electrocoagulation, supplemented by insertion of radium if necessary. Fractional dosage of roentgen rays is the most effective form of radiation therapy for cancer of the larynx, but the results are still too uncertain to justify its use in place of surgical procedures if surgical removal is possible.

As the result of a review of a series of 15 cases of carcinoma of the larynx Hammond [51] concluded that biopsy remained the one indisputable diagnostic procedure in all of the cases. Grading of the malignancy by Broders' method was of value in determining the type of treatment to be given. Eighty per cent of the cancers were of the well differentiated types, either grade 1 or 2. Twenty per cent were anaplastic, grades 3 or 4. Surgical intervention, either by laryngofissure or by laryngectomy, is the procedure of choice in the treatment of intrinsic carcinoma of the larynx. Laryngofissure is particularly adapted to the removal of cordal lesions in an early stage, regardless of the grade of malignancy if the vocal cords still are freely movable. Laryngectomy is particularly adapted to the removal of intrinsic lesions of a low grade of malignancy which are in an advanced stage if there is fixation of the vocal cords. Irradiation is the procedure of choice in the treatment of extrinsic carcinoma of the larynx, particularly of that of a high grade of malignancy which has metastasized to the lymph nodes.

In an excellent article on the physical rehabilitation of the patient who has undergone laryngectomy, Morrison [52] said that the way to solve the worst of the problems which confront the patient and to cure the psychic depression is to get the patient busy at the earliest practicable moment in an endeavor to learn to

48. Fletcher, G. W.: Tumors of the Larynx, Canad. M. A. J. **46**:474-477 (May) 1942.

49. Lenz, M.: Causes of Failure of Roentgen Therapy in Cancer of the Larynx, Am. J. Roentgenol. **46**:21-29 (July) 1941.

50. Figi, F. A.: Treatment of Cancer of the Larynx with Emphasis on Conservatism, Ann. Otol., Rhin. & Laryng. **50**:723-734 (Sept.) 1941.

51. Hammond, A. E.: Diagnosis and Treatment of Cancer of the Larynx, with a Statistical Review of Fifteen Cases, Arch. Otolaryng. **35**:1-29 (Jan.) 1942.

52. Morrison, W.: Physical Rehabilitation of the Laryngectomized Patient, Arch. Otolaryng. **34**:1101-1112 (Dec.) 1941.

talk again by developing a pseudovoice. This at once gives the patient a new aim in life and holds out the hope that if he works steadily and patiently he will soon be able to return to normal life with family and friends and resume his work. Such a return to usual activities is also surely the best antidote for the fear of a return of the malignant lesion. A patient who has undergone laryngectomy meets the total loss of voice in one or more of several ways. He may depend entirely on paper and pencil and may make no effort to talk. He may develop a pseudowhisper, which is produced by exaggerated movements of articulation by using the small amount of air normally present within the mouth. This pseudowhisper is tiring to produce and difficult to understand. The third solution is the use of one of the several forms of artificial larynx. The fourth method of overcoming the total aphonia is to learn the use of a pseudovoice. This voice is produced with the patient's own organs and consists of all the vowel and consonant sounds except the aspirant sound "h." It is perfectly intelligible to any one at any time, never gets out of order because of mechanical failure and sounds exactly like the rather hoarse and guttural speech of a patient who has acute laryngitis. The author said it is by long odds the method of choice and should always be striven for by patients who have undergone laryngectomy. The pseudovoice is produced by aspirating and swallowing air into the hypopharynx and esophagus, from which it is brought out again, at least in part, by voluntary control. This produces a belching sound by setting into coarse vibration some portion of the walls of the hypopharynx, which acts as a pseudoglottis. This guttural sound is changed by the articulating action of the tongue, palate, teeth, cheeks and lips into the sounds of speech. There are thus three parts of the apparatus: an air reservoir in the upper part of the alimentary tract, a pseudoglottis and the articulating mechanism of ordinary speech.

In reviewing a series of approximately 1,100 cases of malignant disease of the larynx, Havens and Parkhill [53] have found that for each 44 cases of squamous cell carcinoma there was 1 case in which some other type of malignant neoplasm was present. They said that although the treatment of other types of cancer of the larynx should follow the same general principles as those laid down for the treatment of carcinoma, there are certain important points of difference to be borne in mind. Sarcoma of the larynx is likely to be found amenable to treatment by conservative operation and to offer a good prognosis. Adenocarcinoma of the larynx may become very extensive before it is recognized but may be curable by radical surgical treatment in spite of this. Hemangioendothelioma responds well to removal and to irradiation of the site of the lesion. Melanoepithelioma and myeloma of the larynx may be classified as medical curiosities. The former is likely to be incurable, but the latter should respond well to appropriate treatment.

A case of leiomyosarcoma of the larynx was reported by Frank.[54] The patient was a woman 20 years of age. The tumor was removed by direct laryngoscopy with bite forceps. The small portion which escaped removal at the time of the first operation was later removed by mirror laryngoscopy. The patient had no recurrence in eighteen months. Approximately 1 per cent of all malignant growths of the larynx are diagnosed as sarcoma; leiomyosarcoma is exceedingly rare. Radiation had no effect on the portion of the tumor which was left in the larynx after the first operation.

53. Havens, F. Z., and Parkhill, E. M.: Tumors of the Larynx Other than Squamous Cell Epithelioma, Arch. Otolaryng. **34**:1113-1122 (Dec.) 1941.

54. Frank, D. I.: Leiomyosarcoma: Report of a Case, Arch. Otolaryng. **34**:493-500 (Sept.) 1941.

Of approximately 1,300 malignant laryngeal tumors seen at the Mayo Clinic in thirty years, only 6 were diagnosed as adenocarcinoma. New and Erich [55] made a comprehensive review of cases of adenocarcinoma of the larynx and reported 1 case in detail. Of the 6 tumors found to be adenocarcinoma, 5 were of low grade malignancy and 1 was classed as grade 4. The authors said that foremost among the difficulties which beset the laryngologist in the diagnosis of adenocarcinoma are a lack of distinctive symptoms produced by the growth and a want of a distinguishable characteristic in the gross appearance. The type of adenocarcinoma most frequently encountered in the larynx is a low grade, slow-growing neoplasm, usually of the mixed tumor type. In the experience of these authors, adenocarcinoma has a tendency to arise in or near the subglottic region. Adenocarcinoma of the larynx appears as firm, smooth thickenings or, more commonly, as a lobular or nodular tumor; it is usually not ulcerated. In general, the prognosis in cases of laryngeal adenocarcinoma should be guarded. In the majority of instances the treatment should consist of surgical procedures of a radical nature. If the lesion is not too extensive, it can be removed successfully by thyrotomy (laryngofissure) in which the growth is excised and its base thoroughly destroyed with surgical diathermy. However, when a considerable portion of the larynx is involved by the tumor, laryngectomy becomes essential. Owing to the site and seriousness of the majority of such growths, one should not rely on intralaryngeal methods (direct or suspension laryngoscopy) to effect a cure. Unless the neoplasm is highly malignant, radiation is of little or no value in the treatment except as a palliative measure in cases in which the growth is hopelessly inoperable.

TUMORS OF THE TRACHEA

Cid and Lopez Bonilla [56] reported a case of giant cell tumor of the trachea. The patient was a man 25 years of age. Bronchoscopic examination and biopsy were performed. Violent hemoptysis immediately followed the removal of the specimen, and the patient died the following morning. At necropsy, when the trachea was opened longitudinally along its posterior surface, a piriform tumor, which measured 2 by 5 by 4 cm., was found at the level of the bifurcation. The tumor rested on the carina in such a manner that its most acute extremity was directed toward the right bronchus, into which it penetrated for a short distance. Distal to the tumor was a blood clot which obstructed the lumen of the bronchus. The blunt extremity of the tumor passed the midline of the trachea on the left side and seemed to cause a moderate degree of obstruction of the left bronchus. Microscopic examination revealed a giant cell tumor. The authors said that the extraordinary rarity of such a tumor makes the case one of special interest. A search of the literature indicated that this is the first time that a giant cell tumor of the trachea has been reported.

TUMORS OF THE ESOPHAGUS

Samson and Zelman [57] reported a case of pedunculated tumor of the esophagus in a white man 70 years of age. The tumor had been regurgitated on several occasions; at times this had caused interference with respiration. The polyp was

55. New, G. B., and Erich, J. B.: Adenocarcinoma of the Larynx, Ann. Otol., Rhin. & Laryng. **50**:706-714 (Sept.) 1941.

56. Cid, J. M., and Lopez Bonilla, J., Jr.: Giant Cell Tumor of the Trachea, J. Thoracic Surg. **11**:210-215 (Dec.) 1941.

57. Samson, P. C., and Zelman, J.: Pedunculated Tumors of the Esophagus, Arch. Otolaryng. **36**:203-211 (Aug.) 1942.

extirpated through an esophageal speculum. The postoperative course was uneventful. Microscopic examination revealed a fibroepithelial polyp. The authors also reviewed a series of 25 similar cases which had been recorded in the literature. In 8 of these cases cure was reported after removal of the tumor, by external esophagotomy in 2 cases and by endoscopic means in 6. After thorough consideration of both methods of treatment it was the authors' opinion that endoscopic removal is superior to extirpation by external esophagotomy.

A case of fibroma of the esophagus was reported by Lahey.[58] The patient was a woman 48 years of age. The tumor was removed surgically, and the patient made an uneventful recovery. This case was reported because of the difficulty in distinguishing the lesion from an intrathoracic goiter and because it demonstrates plainly that when the recurrent laryngeal nerve and the inferior thyroid artery are anterior to the tumor the tumor is not of thyroidal origin but often of esophageal origin.

Dickes, Knudsen and Franco [59] reported a case of massive recurrent hematemesis in a girl 15 years of age. The condition apparently began as hepatitis with splenomegaly and was temporarily controlled by splenectomy. Although the liver showed no evidence of permanent damage and there was no increase in portal pressure, the hemorrhages recurred. Esophagoscopy ultimately revealed the presence of polyps, which were removed by fulguration. The patient recovered.

58. Lahey, F. H.: Esophageal Tumor: Report of a Case, Lahey Clin. Bull. **2**:194-197 (Jan.) 1942.

59. Dickes, R.; Knudsen, A. F., and Franco, S. C.: Multiple Polyps of the Esophagus: Report of a Case with Complicating Recurrent Gastrointestinal Hemorrhages, Arch. Int. Med. **70**:121-131 (July) 1942.

Abstracts from Current Literature

Ear

DEFICIENCIES IN OTOLOGIC SURGERY. FRANCIS I. LEDERER, New York State J. Med. **42**:2033 (Nov. 1) 1942.

The author states that present day specialists have come to disregard certain basic principles, have made obeisance to robots and have depended too much on manual dexterity, chemotherapy and "science." They sometimes have wandered far away from basic anatomy, physiology and pathology, have parted with tradition and have neglected the contributions of their predecessors. On the other hand, some have taken a courageous initiative and have ventured new thoughts and offered new surgical technics. But, in the words of Jacobi, "There are many operators, but few surgeons." Five otosurgical necessities are discussed: (1) basic knowledge, which must include the anatomy, physiology and pathology; (2) surgical judgment; (3) surgical skill; (4) proper medical and surgical management, and (5) after-care.

The paper is an attempt to evaluate the "results" of otologic knowledge as it has developed over the past half-century, to point out the reasons for error and defeat and to profit by the experiences of otologists who have set these experiences down as a guide for their successors.

VOORHEES, New York.

THE EVALUATION OF THE AUDIOMETER IN TESTING HEARING. C. C. SWANN, South. M. J. **35**:280 (March) 1942.

The author states that the audiometer enables otologists to determine much more accurately than by other means the diagnosis and scientific treatment of hearing defects and that, as a universally accepted method of testing, it is invaluable in that it makes possible accurate plotting of a curve for the quantitative loss of hearing for different frequencies and provides a check on variations in hearing while treatment is being carried out. Other types of tests are discussed in some detail. The importance of tests with the spoken and whispered voice and with tuning forks is stressed. Tuning forks are especially valuable for testing of low tones, for comparison of air and bone conduction and for estimation of the condition of the conductive apparatus.

The author emphasizes the importance of making more than one audiometric test and calls attention to the fact that patients with nerve deafness are fairly constant in their responses, while patients with obstructive deafness often show considerable variation in their audiograms from week to week.

The audiometer is of great value in making mass hearing tests on school children, but these tests are often inaccurate, and children whose hearing appears impaired after such a test should be referred to an otologist for final diagnosis and advice. Several audiograms, illustrating various types of deafness, are shown.

SINGLETON, Dallas, Texas.

INFLUENCE OF VARIATIONS IN THE SIZE AND STRUCTURE OF THE CRANIAL VENOUS SINUSES ON THE CLINICAL PICTURE OF SINUS THROMBO-PHLEBITIS. ERNEST M. SEYDELL, South. M. J. **35**:555 (June) 1942.

Seydell calls attention to the fact that the variations in the size and the structure of the cranial venous sinuses have not received the attention they deserve. He records Woodhill's findings in reference to such variations, based on a study of 100 dural preparations. Seydell states that these variations have a direct bearing on certain clinical phases of otitic sepsis. If thrombosis occurs in a lateral sinus that carries the main portion of the return blood from the brain, such symptoms as headache, nausea, vomiting, slow pulse, photophobia, choked disk, ocular palsy, aphasia, indifference, torpor and coma may result. In some patients it is impossible to differentiate the symptoms which result from increased intracranial pressure due to disturbed cerebral venous circulation from those which accompany abscess of the brain, meningitis or otitic hydrocephalus.

The relationship between these variations and the contradictory findings encountered in making the Tobey-Ayer test is discussed in considerable detail. The author states that abnormalities of the lateral sinus can be determined by roentgen examination of the jugular foramen and advises that such an examination be made in all cases of otitis media and mastoiditis with symptoms suggesting involvement of the intracranial sinuses.

The article contains thirteen references.

SINGLETON, Dallas, Texas.

ACUTE OTITIS MEDIA AND MASTOIDITIS. ERNEST M. SEYDELL, South. M. J. **35**:658 (July) 1942.

Seydell states that some of the factors that decide whether suppurative otitis media will disappear after paracentesis or whether it will later be necessary to perform a mastoid operation are (1) the virulence and the type of the infecting organism, (2) the anatomic structure of the mastoid process, whether diploic or pneumatic, and (3) the physical condition of the patient and his own constitutional peculiarity. He then discusses in some detail the symptoms of fever, pain, discharge and sensitivity of the mastoid to pressure as related to acute otitis media and mastoiditis.

Persistent or recurrent fever in the presence of middle ear infection, severe pain in the ear or over the mastoid process which continues after paracentesis, discharge that continues or increases or after becoming scanty again becomes profuse, and tenderness on pressure over the mastoid process that does not recede after the acute phase or tenderness that recurs after having subsided are indications of mastoidal infection.

Eight indications for operation on the mastoid process are listed, with the observation that this is usually imperative in the presence of any one or more of the indications named. Treatment is discussed under the various headings of local therapy, general therapy, sulfonamide therapy, and operative therapy.

SINGLETON, Dallas, Texas.

Nose

VITAMINS FOR THE PREVENTION OF COLDS. DONALD W. COWAN, HAROLD S. DIEHL and A. B. BAKER, J. A. M. A. **120**:1268 (Dec. 19) 1942.

The authors conducted two series of studies on the value of large doses of vitamin C alone (1939-1940 series) and of large doses of mixed vitamins (1940-1941 series) in the prevention of colds.

Students of the University of Minnesota volunteered, just as in the previous studies on cold. Careful records were kept of each student with regard to the condition of the upper respiratory tract and the dose of vitamins given. If any one had a cold, he was to report the severity thereof and the speed of recovery. One group received vitamin C alone, and another group serving as a control received placebo tablets of the same size, shape, appearance and taste as the ascorbic acid tablets. The second series of studies dealt with the administration of multiple vitamins to one group, while another group received placebo capsules.

The authors summarize their final study as follows: "This controlled study yields no indication that either large doses of vitamin C alone or large doses of vitamins A, B_1, B_2 C and D and nicotinic acid have any important effect on the number or severity of infections of the upper respiratory tract when administered to young adults who presumably are already on a reasonably adequate diet."

GORDON, Philadelphia.

LEAD INTOXICATION FROM A BULLET LODGED IN THE SPHENOID SINUS AND SURGICAL REMOVAL OF THE NIDUS. CHARLES E. FUTCH, J. A. M. A. **121**:582 (Feb. 20) 1943.

The author discusses plumbism briefly and reports the case of a man who carried a lead bullet in the left sphenoid sinus for ten years, as the result of the accidental discharge of a revolver. The patient presented symptoms of true lead poisoning for several years; the laboratory evidence, however, was not positive.

The bullet was removed by a submucous resection, after which the patient made an uneventful recovery. No complications were encountered during or after the operation. The consulting neurologist advised administration of large amounts of calcium to fix free lead that may be present in the tissues.

GORDON, Philadelphia.

OSTEOPHYTES OF THE MAXILLARY SINUS. L. W. DEAN JR. and W. H. DIEHL, Laryngoscope **53**:112 (Feb.) 1943.

The authors cite Zuckerkandl and others with regard to the occurrence of osteophytes of the sinuses. Chronic inflammation produces hyperostosis. The new bone thus formed may lie loose in the lining of the sinus, with no attachment to the bony wall. This may occur in any of the sinuses. Several small osteophytes tend to coalesce into a larger one Their operative removal is easy provided the opening into the sinus can be made large enough. Hence, early operation is advisable, before the osteophytes become too large. The authors present 2 cases of osteophyte formation in the maxillary sinus.

HITSCHLER, Philadelphia.

RHINOPHYMA, WITH NEW ETIOLOGIC AND THERAPEUTIC CONSIDERATIONS. MICHAEL M. WOLFE, Laryngoscope **53**:172 (March) 1943.

In the literature of the past twenty years, rhinophyma has been held to be the terminal phase of acne rosacea. The excessive use of alcohol, tea and coffee has been considered causative. There are several weaknesses in these etiologic theories. The author offers some new considerations: 1. The condition may be related to an abnormal output of androgens. Such an output occurs in the male at about the same age as does rhinophyma. 2. At about the same age, some men, for various reasons, are on restricted diets, often low in vitamin C. A deficiency of this vitamin may be an etiologic factor. Reasons for these theories are given.

A case is then presented, with laboratory observations. Koagamin (an aqueous preparation of oxalic and malonic acids) and koaginol (a preparation in oil of the diethyl esters of dicarboxyl acids) were used to reduce bleeding. Vitamin C, which had been low in the diet, was supplied in adequate amounts. The rhinophymatous tissue was pared down with a razor blade. An excellent result was obtained.

HITSCHLER, Philadelphia.

Miscellaneous

HEADACHE AND FACIAL NEURALGIA: CLASSIFICATION AND DIFFERENTIAL DIAGNOSIS. T. ROY GITTINS, Laryngoscope **53**:27 (Jan.) 1943.

Gittins points out the prevalence of the symptom of "pain somewhere in the head or face" and mentions how migraine and other types of vasomotor rhinitis have been unsuccessfully treated and the patients bandied about in the past from one group of specialists to another. Part of the disrepute of sinus surgery is due to its ill advised use in treatment of such headache.

A vasomotor basis for periodic headache or facial neuralgia usually exists, "especially if the symptoms are repeated over fairly long periods with intervals of good health, with or without associated symptoms . . ." and if in such cases the results of physical and laboratory examination or operative observations are without significance. Migraine, allergic headache and other forms appear as types of vasomotor headaches. Patients with such headache have often been wrongly treated and operated on, the nature of the treatment or the operation depending on the particular branch of medicine in which the physician had specialized.

The proper procedure is first to make a careful differential diagnosis. The author discusses how to make such a diagnosis and the importance, when encountered, of the various associated cortical symptoms (scintillating scotoma, paresthesia, nausea and vomiting, vertigo, aphasia and amnesia and the motor symptoms).

He says: "Real pathologic changes in the nose, sinuses, eyes, teeth, brain or any part of the head cannot be responsible for such systematic reproduction of similar explosive phenomena over relatively long periods of time with intervening periods of good health."

Migraine in children is more common than is generally thought. The headache is often overshadowed by other symptoms, such as various gastric disturbances and fever. There may be associated cortical symptoms. By a careful history the presence of prodromal symptoms can often be elicited.

The author gives and discusses a rather complete classification of headache and facial neuralgia for everyday practice. When headache is a symptom incidental to some other condition, treatment should be directed to the cure of the underlying trouble. Headache as the main symptom is of two types: the acquired and the hereditary. The former includes headache from sinal, dental and ophthalmic disease, as well as neuralgia, neuritis and the reflex and vascular syndromes. It is important to rule out such forms of headaches when one is making a diagnosis of migraine, which is of the hereditary type. By so doing many patients will be spared unnecessary treatment and operations.

HITSCHLER, Philadelphia.

LOCAL SULFONAMIDE THERAPY IN OTOLARYNGOLOGY. A. C. FURSTENBERG, Laryngoscope **53**:93 (Feb.) 1943.

The author points out that the local use of sulfanilamide in otolaryngologic practice has not accomplished as much as he first thought. In the light of further experience with it, the effects have been good, but not miraculous. It is an adjunct to, and not a substitute for, good surgical treatment. He emphasizes this point.

In the simple mastoid operation the author fills the wound with the drug, sutures the upper part and merely places a small wick in the lower end of the wound. The wick is left in place until drainage does not have to be maintained. Cultures showed that such a wound becomes sterile in five to ten days. This happens only if nothing is reintroduced into it. The

author disapproves of the older practice of removing and reinserting a pack. Such a pack, cultures show, often contaminates the wound and delays healing. With the newer method described there should be a dry, or nearly dry, middle ear and mastoid cavity in ten days. If the area is not dry by this time, surgical exploration should be made.

A similar postoperative procedure is followed in the radical operation. With the method described there are faster healing and fewer postoperative infections of the wound.

Sulfathiazole is not as satisfactory as sulfanilamide because it tends to cake in the wound.

Evidence has been presented by others to show that sulfanilamide applied locally retards healing. This retardation is more than overcome in the simple mastoidectomy wound by the faster healing due to absence of secondary infection. The clinical fact that a mastoid so treated heals faster proves this. The use of sulfanilamide locally in the radical mastoidectomy wound is particularly favorable when the retardation of growth of granulation tissue by the drug is a distinct advantage, so that epithelization occurs more quickly.

The author states that he has not had much experience with sulfanilamide in other phases of otolaryngologic work, but mentions its value in the preparation of a bed for a skin graft after radical mastoidectomy, surgical procedures on the nose, laryngectomy or tracheotomy.

Certain investigators have not found sulfanilamide useful in septic wounds. Such work must be kept in mind in evaluation of the drug. The author does not approve of its use in aseptic wounds because of the retardation of healing and the production of a tissue reaction.

HITSCHLER, Philadelphia.

AN ARTIST'S APPROACH TO RESTORATIVE PROSTHETICS. BEAVER EDWARDS, Mil. Surgeon **92**:197 (Feb.) 1943.

The materials for use in the making of restorative prostheses have improved in quality, adaptability and durability, through metals, gelatin and wax and due to the use of latex compounds, which are by far the most satisfactory at present. While surgical treatment has the advantage of permanency and is the positive solution in many cases, there are circumstances under which the restorative prothesis, with its attendant avoidance of the debilitating effects of operation, loss of time from employment and monetary expense, would seem the wiser choice. A well schooled artist in the field of prosthetics, trained through years of study and observation to detect falseness and variation from the natural, should be able to accomplish splendid results in collaboration with the surgeon. The author presents photographs of various restorations, and one is struck by their beauty and naturalness.

H. MILLER, Philadelphia.

IRRADIATION IN INFECTIONS. LEONARD B. GOLDMAN, New York State J. Med. **42**:1341 (July 15) 1942.

Goldman studied 640 cases of infections of all types at the Queens General Hospital over four and one-half years. Many observers, notably Schillinger, have noticed improvement in cases of otitis and mastoiditis after diagnostic exposure to roentgen rays. But the bacteria were not destroyed, merely rendered less virulent. Furuncles (in the ear) may be aborted by irradiation if this is done early. Relief of pain is immediate. Irradiation for bronchiectasis diminishes the expectoration and relieves cough. Parotitis as a complication of debilitation is often of sufficient gravity to cause death. Irradiation is a specific therapeutic measure for this serious condition. In the case of adenitis the course is shortened and the incidence of suppuration is lowered. The author's assertion that "when roentgen therapy is used in infections, surgery is seldom necessary" seems an overstatement. Nothing replaces surgical treatment when an acute infection holds a life in the balance. Furthermore, delays due to too much dependence on radiation or other mechanical agents may be dangerous indeed. The advantages of radiation therapy are a lower fatality rate, earlier recovery, fewer complictions and less scarring and deformity. Discomfort and pain are diminished shortly after institution of therapy; the stay in the hospital is shortened, and there are fewer recurrences of the infection.

VOORHEES, New York.

INTERNAL WIRE FIXATION OF FACIAL FRACTURES. WILLIAM M. ADAMS, Surgery **12**:535 (Oct.) 1942.

After the use of numerous extraoral appliances, including those attached to plaster head caps, for immobilization of fractures of the maxilla and other facial bones, the author has found that these appliances are complicated, their preparation and application are time consuming, they are cumbersome and uncomfortable for the patient and they require close watching and repeated adjustment.

In carrying out the author's method, an incision is made over the infraorbital ridge. A hole of sufficient size to accommodate a no. 25 stainless steel wire is bored through the infraorbital ridge. The wire is then threaded through the opening and looped over the ridge, and both ends are passed together along the anterior wall of the antrum into the upper sulcus over the second molar teeth. The fractured bone is then elevated to its normal position, and the wires are drawn taut and fixed to one or more of the teeth. In cases of fracture of the maxilla associated with fracture of the infraorbital ridge it is sometimes necessary to immobilize the major fragment of the ridge to the outer half. This may be done through the same incision. The wires are removed in three to nine weeks. The procedure requires a minimum of time and equipment and eliminates the head cap and external appliances formerly used.

H. MILLER, Philadelphia.

EXTENSIVE, MUTILATING FACIAL DEFECT; COSMETIC CORRECTION WITH LATEX MASK. ADOLPH M. BROWN, Surgery **12**:957 (Dec.) 1942.

Constitutional diseases, such as lupus, leprosy and syphilis, often make plastic surgery, as a method of reconstructing missing parts of the body, unwise. A case in which extensive destruction of the face occurred is presented to demonstrate that a large latex compound prosthesis can be constructed and adapted to conceal unsightly, and even open, lesions. It also indicates that a prosthesis of latex may be designed to cover moving parts, with comfort and esthetic satisfaction to the patient. The latex mask over the hideously disfigured face produced an astonishing transformation. The patient, after years of idleness and despair, has now been given a light, part time occupation and is happy, and even proud of his prosthesis.

H. MILLER, Philadelphia.

OCULOLARYNGOPHARYNGOVELOPALATIVE MYOCLONIA IN A BULBOPONTILE SYNDROME. C. JAKOB and JUAN C. MONTANARO, Rev. neurol. de Buenos Aires **7**:85 (April-June) 1942.

Jakob and Montanaro give an account of a 35 year old man who suddenly was taken with a pain at the left angle of the mouth, dizziness and vomiting. Peripheral paralysis of the left side of the face and paresis of the right limbs developed rapidly. The patient was syphilitic. Examination revealed paresis of the left sixth nerve and paralysis of the left fifth and seventh nerves, right hemiparesis, anesthesia of the left side of the face and right hemihypesthesia. In addition, there was myoclonia of the palatal, pharyngeal, laryngeal and ocular muscles. He died about three years after the attack. Careful examination of the brain revealed a focus of degeneration in the left side of the tegmentum involving the nuclei of the cranial nerves from the fifth to the eighth. The fifth and seventh nerves were completely interrupted; the eighth, only partly. The following pathways were involved: the median and lateral fillets, the tract of Gowers, the central tegmental tract, the middle cerebellar peduncle and the greater part of the left reticular formation. The temporopontile and the pyramidal tracts were partially destroyed. In addition, the nuclei of the reticular formation, the superior olive, the cerebellar root of the vestibular nerve, the floccular peduncle in part and the mesencephalic root of the trigeminal nerve were destroyed. The authors conclude that velopalatine myoclonia is not necessarily due to a circumscribed focal lesion but is the functional result of an alteration of the total equilibrium of paleodentato-olivary systems.

BAILEY, Chicago. [ARCH. NEUROL. & PSYCHIAT.]

SUPPURATIVE OTOGENIC AND RHINOGENIC MENINGITIS WITH AND WITHOUT SULFANILAMIDE THERAPY, WITH PARTICULAR REFERENCE TO PNEUMOCOCCIC MENINGITIS (TYPE III). FRANZ ESCHER, Pract. oto-rhino-laryng. **4**:23, 1942.

The author reports 2 cases of meningitis, the disease in the first case being of otogenous origin and in the second case the result of a suppurative process in the sphenoid sinus. The meningitis in the first case was due to Streptococcus haemolyticus, while in the second case the offending agent was the pneumococcus type III. Cure was effected in both cases with intensive sulfanilamide therapy.

The author states that prior to the advent of sulfanilamide therapy the only hope of an occasional cure occurred in the cases of otitic origin in which the spinal fluid remained sterile. On the other hand, if the meningitis was rhinogenic, even though the spinal fluid remained sterile for a time, the prognosis was much graver. Greater hope can now be felt, particularly if early thorough elimination of the infective process has been carried out, together with the simultaneous administration of intensive chemotherapy.

Escher stresses the fact that in three fourths of his cases of otitic meningitis the pneumococcus type III was the causative organism and the condition was invariably associated with the usual complications, such as extradural abscess, sinus thrombosis and petrositis. Finally, he stresses the importance of secondary sources of suppuration in the sinuses, particularly the sphenoid, which may, either alone or in conjunction with otitic foci, produce meningitis.

PERSKY, Philadelphia.

Society Transactions

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

JAMES A. BABBITT, M.D., *President*

E. W. HAGENS, M.D., *Editor of Abstracts*

Forty-Eighth Annual Meeting, Atlantic City, N. J., June 1 to 3, 1942

The Nasopharynx. DR. GEORGE M. COATES, Philadelphia.

Careful, adequate examination of the nasopharynx is important and very worth while. If necessary, local anesthesia and even sedation should be used. When a mirror cannot be employed, the Yankauer speculum should be tried. With young children and certain adults, finger palpation of the nasopharynx gives important information. The various forms of pathologic change in this area are discussed and the treatment indicated.

DISCUSSION

DR. JOHN R. HONISS, Rochester, N. Y.: The lack of information on this area is due, I believe, as Dr. Coates stated, to difficulties in the examination and, may I add, also in the deductions from this examination. One cannot always see trouble or infection or abnormalities. I am making a special plea for finger exploration. The appreciation of conditions present that one may get from the sense of touch and the feeling of the tissues is valuable, and this information is enhanced by repeated palpations and practice. One may feel neoplasms, adhesions and loose lymphoid tissue, also the condition of the eustachian orifice and cushions.

Mirrors, retractors and scopes will not give a good idea of firmness or looseness of the tissues.

DR. WALTER H. THEOBALD, Chicago: This epipharyngeal space may be called the overlooked area, because its importance is not sufficiently considered or emphasized in determining the sources of many clinical conditions and foci of infection. I wish to impress on you that such conditions as recurrent colds and sore throats, postnasal secretion, chronic cough and occasional bleeding find their origin in lymphoid hypertrophy, retention crypts or true Thornwaldt's disease. In my own experience, systemic conditions such as arthritis, fever and even asthma have been explained on the basis of inflammation in this area.

DR. THOMAS E. CARMODY, Denver: I want to speak especially of the Yankauer speculum. This instrument can be used without an anesthetic. I always used to anesthetize the locale, and some of the patients objected more to the local anesthesia than they did to the examination with the pharyngoscope.

Children, of course, should probably in most cases be given an anesthetic.

In the treatment of any nasopharyngeal condition the area should be cleaned first with an alkaline solution or a preparation of 4 parts alkaline solution with about 1 part of hydrogen peroxide. It loosens the secretions much better.

DR. CHARLES D. BLASSINGAME, Memphis, Tenn.: Adenoidectomy requires deeper anesthesia than tonsillectomy. The palate must be relaxed. The mouth must be wide open and the chin extended. In that position the Yankauer speculum can be employed with much greater advantage.

In regard to the removal of the remaining tissue after use of the LaForce adenotome, I do not believe that using the finger cot or gauze is good technic. With a punch forceps as large as can be got through the Yankauer speculum—the larger the better—one can peel off all adenoid tissue down to the muscle and aponeurosis, especially in the deeper part of the fossa.

Otolaryngologic Problems of Aviation in World War II. MAJOR PAUL A. CAMPBELL, Randolph Field, Texas.

Aviation is placing considerable strain on the ears of the young men in this branch of the service. Fortunately most of the changes are transitory. In aero-otitis myringotomy is contraindicated. Heat should be applied, analgesic drugs should be given for relief of pain, and shrinkage of the orifice of the eustachian tube should be done. At present there is no single

simple remedy for air sickness. Aerosinusitis has increased in frequency as greater altitudes and speeds of ascent have been reached. The mechanism of its production is similar to that of aero-otitis. Those with acute sinusitis or rhinitis should not fly. The effect of anoxia on hearing is a problem, but up to the present no definite conclusions have been reached as to a definite audiometric pattern.

DISCUSSION

DR. EUGENE R. LEWIS, Los Angeles: I am interested in certain facts which have to do with the clouded history of the development of aviation medicine.

I take it that my part in today's discussion stems from paragraph 113, S. O. 243, A. G. O., Oct. 18, 1917, "assigning to duty as members of a Medical Research Board, Major John B. Watson, S.O.R.C., Major Eugene R. Lewis, M.R.C., Major William H. Wilmer, M.R.C., Major Edward G. Seibert, M.R.C." Dr. Yandell Henderson was added to this board in civilian capacity and was constituted chairman. This organization was unchanged during the following sixteen months and to it were delegated the following powers:

1. To investigate all conditions which affect the efficiency of pilots.
2. To institute and carry out, at flying schools or elsewhere, such experiments and tests as will determine the ability of pilots to fly in high altitudes.
3. To carry out experiments and tests, at flying schools or elsewhere, to provide suitable apparatus for the supply of oxygen to pilots in high altitudes.
4. To act as a standing medical board for the consideration of all matters pertaining to the physical fitness of pilots.

DR. ROBERT J. HUNTER, Philadelphia: I was interested in hearing what Major Campbell had to say about the question of impairment of hearing resulting from a noise level of the plane and the question of the changing of the voice.

In connection with this measurement of the noise level associated with dizzy spells, I am wondering what the ordinary noise of one's environment is. There are some statistics on that, but I am not acquainted with them. Every one is normally subjected to a certain pressure on the ear drums at that noise level and any one who steps into a sound-proof room can demonstrate that to himself at once. When one goes into a sound-proof room there is immediately a sense of pressure on the ear drums. That sense of pressure is very similar to what is felt in going to a different altitude or with a change of barometric pressure. When one dashes down into the subway in New York one has a similar feeling, which, of course, is not due to barometric change but to absence of the noise to which one is accustomed.

It occurred to me that some of the sensations the men get from the greatly increased noises to which they are subjected in the airplanes may be due to the physical sensation of pressure just described in addition to the actual noise.

I wonder whether some of those voice effects at high altitude could be indirectly due to the altitude but directly due to the change of hearing that occurs. Every one is acquainted with the defect of hearing which causes the patient to raise his voice, and I am wondering whether some of these changes at altitudes are due to the effort of the pilot to hear his own voice.

Another thought that occurred to me was the question of the changes of pressure between the middle ear cavity and the passage leading to the drum membrane. I am wondering whether a tight-fitting helmet would lighten some of the pressure effect; that is, if the helmet fitted tightly, the air inside the external cavity would have a differential pressure in comparison with that at the altitude to which the pilot was flying.

Major Campbell mentioned the fact that in some of the patients aerotitis disappears in three days and in others it continues for a longer period, possibly twenty-one days.

It occurred to me that the difference in the period of getting well may be due to a slight difference in pathologic condition. There may be two classes of cases. In the case that he showed you, with the hematoma, there was a little ecchymosis in the ear drum. On the other hand, one sees some cases in which the middle ear is filled with blood. And it occurred to me—I should like to ask the Major's opinion on this—that if the hemorrhage occurred in a small vessel in the middle ear so that the blood flowed out into the middle ear cavity and thus lessened the pressure by filling that vacuum, one would have a blood-filled cavity without trauma of the drum.

I have seen a man who after an airplane flight had a drum full of blood, and one would have thought it would take a long time for the ear to get well, but in forty-eight hours that ear was well.

MAJOR PAUL A. CAMPBELL, Randolph Field, Texas: With regard to Dr. Hunter's statements, my associates and I have made studies of the physical action of altitude on the voice

and have listed recordings by means of an electro-oscillograph. We are certain that there is a difference in frequency between the voice at a high altitude and the voice on earth. We made the measurements on exactly the same voice.

As far as tight-fitting helmets and earphones are concerned, all helmets today, I believe, have a ventilation slit by which pressures may be equalized on ascent and descent.

Concerning Dr. Hunter's question as to the reason why a hematoma in the middle ear clears up so rapidly, I believe that is entirely due to the fact that the pressure is immediately equalized by the hemorrhage in the middle ear. As soon as the pressure is equalized, there is no longer so-called barrel trauma, and the course which follows is entirely related to the speed by which the ear can be emptied of the bloody contents; whereas, if that equalization does not take place, there is even in mild cases of aerotitis a traumatic influence at all times, so that it may take as long as ten days, or more, for the equalization to take place, and recovery cannot take place without the equalization.

Practical Points in Diagnosis and Treatment of Sinusitis. DR. W. C. BOWERS, New York.

This article was published in full in the ARCHIVES OF OTOLARYNGOLOGY (**36**:327 [Sept.] 1942).

On Planning Nasal Surgery. DR. ARTHUR W. PROETZ, St. Louis.

This article was published in full in the ARCHIVES OF OTOLARYNGOLOGY (**37**:502 [April] 1943).

DISCUSSION

DR. GEORGE L. WHELEN, Philadelphia: Not infrequently the situation which Dr. Proetz describes as too much and too frequent surgical intervention is due entirely to the fact that the patient and his medical advisers suspect that because he did not obtain relief from the previous surgical intervention that intervention was insufficient or was incompletely accomplished. More surgical treatment is advised, or another rhinologist is consulted. This situation, I believe, could be overcome if rhinologists would conscientiously arrive at conclusions based on pooled statistics and experience as to the types of surgical procedures which are indicated in certain thoracic and other conditions. Much of the effect of improper surgical treatment and its attendant criticism could in this way be avoided.

Occasionally an internist may not be able to detect the cause of a patient's thoracic symptoms. He may therefore suggest that the focus of infection is in the nose and he may expect the rhinologist to change a diseased nose into an ideal anatomic and physiologic specimen. Even if this were possible, the patient's symptoms might be totally unaffected. Such practitioners usually belong to the group of enthusiasts, not too well informed, who are all too ready to promulgate the idea of cause and effect between the nasal sinuses and other organs, usually those in the chest.

As to operations on chronically diseased sinuses, my feeling is that with the intranasal surgical approach it is not possible for me to do as satisfactory work as with the external approach. Operations on the sinuses are difficult at best. When they are done without visualizing the field on which one is operating, one may expect poor results. For this reason I prefer to operate by the external route when opening the frontal or the maxillary sinuses or the ethmoid cells, when dealing with osteomyelitis or with orbital abscess or when operating secondarily for ethmoid complications.

It would seem that the Caldwell-Luc operation is the most satisfactory of all operations for the treatment of the nasal accessory sinuses and the one which should yield the highest percentage of good results.

DR. GORDON D. HOOPLE, Syracuse: The idea of planning for a nasal operation appeals to me. Planning prohibits the mere statement "This requires a resection of such and such a part" and calls for additional information as to why and how the resection should be performed.

With acute frontal sinusitis which demands surgical relief, I believe it is better to do the simple Hajek frontal operation, making an external opening into the sinus and establishing drainage with a rubber tube, than to manipulate intranasally in the region of the nasofrontal duct. (I use the word manipulate advisedly.) When the infection subsides, if the external approach has been used, the duct is saved and is free to resume normal function. Incidentally, a simple test tells whether the duct is open and functioning again: Some sterile saline solution is instilled through the drainage tube; if the patient reports that fluid is collecting in the throat, one knows that the duct is patent and that the drainage tube can be removed and the wound allowed to heal.

Neurologic Lesions Which Influence the Sense of Smell. Dr. A. C. Furstenberg, Dr. Elizabeth Crosby and Dr. Brown Farrior, Ann Arbor, Mich.

Consider the patient who tells his physician that he detects an odor which is evanescent but is present often enough and of sufficient intensity to cause him great annoyance. It frequently appears at times that are embarrissing to him, at which he suspects he is obnoxious to his friends. If an organic lesion is responsible for this symptom, it is probably one in the upper air passages giving rise to odorous gases which escape at irregular intervals. For example, chronic maxillary sinusitis, particularly that of dental origin, may empty noxious contents at varying intervals into the nose and give rise to the olfactory disturbance to which we refer. The intermittent spilling of odoriferous pus into the nasal cavities stimulates the olfactory nerves and gives rise to symptoms for which the patient seeks relief. Or the emanation of gases from infective lesions about the teeth, frequently facilitated by the sucking action of the mouth, may give rise to odors of a putrid nature that are most offensive to the patient.

The physiology of olfactory fatigue is well understood. The sustained presence of the pungent or noxious gas overtaxes the olfactory mechanism to the point of exhaustion with the result that the patient ultimately loses all consciousness of its presence.

In describing organic lesions which are the cause of unpleasant olfactory perceptions of a fleeting character, one must not fail to include lesions of the uncinate convolution, in which are found the olfactory association centers. The patient complains of a perverted sense of smell, usually of some foul odor, and exhibits a definite seizure to which the term "uncinate attacks" or "uncinate fits" has been given. The symptom of an unpleasant odor is in the nature of a hallucination and is associated with other signs that clearly identify the disease.

The otolaryngologist sees a great many patients who are suffering from illnesses of psychologic origin.

Every one is thoroughly acquainted with the local lesions which produce anosmia, such as atrophic rhinitis, allergic reactions, nasal polypi, neoplasms, and chronic infective granulomas, and understands that their injurious effects are felt directly on the terminations of the olfactory nerves in the epithelium of the olfactory region high in the lateral wall of the nose and in a corresponding portion of the nasal septum. There occur a number of well defined central lesions that produce disturbances of the olfactory and gustatory senses which come to the attention of the rhinologist from time to time and which are important enough from the point of view of his professional standing and his joy of practice to be better understood. Each is a well defined clinical entity arising as a result of a pathologic change within the olfactory mechanism and giving rise to a symptom complex which clearly indicates its anatomic location. An interest in these lesions provides an opportunity and an incentive to improve one's knowledge of anatomic relationships even though it is motivated largely by a curiosity for information of a practical value.

Meningioma of the olfactory groove presents a characteristic symptom complex consisting of anosmia, papilledema and disturbances in the sensorium.

Meningioma of the sphenoidal ridge is characterized by a symptom complex which includes usually unilateral anosmia or hyposmia as a late manifestation of the disease. Frequently the early signs of this lesion are unilateral proptosis with impairment of vision. Examination of the fundus reveals homolateral optic neuritis with a central scotoma, after which optic atrophy appears.

It is estimated that approximately 20 per cent of the tumors of the temporal lobe produce some form of olfactory disturbance. This is most frequently characterized by olfactory hallucinations, a significant symptom of the so-called uncinate fits or attacks. These indicate an irritative lesion somewhere within the region of the uncinate lobe. The attacks are characterized by "an evil smell" or "an evil taste." The patient may experience a mild epileptiform seizure in which he is dazed but does not necessarily lose consciousness.

DISCUSSION

Dr. Arthur W. Proetz, St. Louis: When a patient comes complaining of an impairment of the sense of smell, his sense of smell is practically gone. Sometimes by means of olfactory tests one can find a patient whose sense of smell is not acute, but he is not aware of it unless the sense is practically gone. So one finds it rather difficult to use qualitative tests for the localization of these lesions.

Furthermore, the end organ of the sense of smell is a very simple thing, and the central connections, as Dr. Furstenberg has shown you, are very complex. Shaeffer enumerated some twenty-four structures that are involved in the distribution of the perception of smell.

I was much interested in Dr. Furstenberg's demonstration of the regions in which lesions must occur in order to have an effect on the sense of smell. You will notice that the diag-

nosis in no case was made from any observation of the sense of smell but was made from roentgen evidence and other neurologic signs. Actually, the sense of smell helps little.

When a patient comes complaining of a constant impression of an odor, one can be sure it is not peripheral, because the fatigue element enters early and obviates perception of a peripheral odor.

Dr. French K. Hansel, St. Louis: Probably one of the most common causes of the loss of the sense of smell is the edema of the olfactory area seen in allergy. My associates and I see many of these patients who complain of a loss of the sense of smell. When we question them we find that they can detect certain flavors of food, indicating that there is not a complete loss. It is amazing how long a patient can go with a marked reduction in the sense of smell and yet come back to normal under proper management.

In Dr. Furstenberg's discussion regarding central lesions, the point was not brought up as to whether the senses of smell and taste are unilateral or bilateral in the temporal lobe. Dr. Sachs of St. Louis reported in his book on brain surgery that he had had 2 cases in which tumor of the left temporal lobe completely destroyed the senses of taste and smell.

Dr. Frank R. Spencer, Boulder, Colo.: I think of loss of the sense of smell more as a symptom than as a disease, just as I think of asthma as a symptom complex. It is not a true disease in itself. One must find the cause of the symptom or of the group of symptoms.

For practical clinical purposes and for teaching, I like to classify the neurologic conditions causing this symptom as follows:

1. Benign and malignant tumors of the brain which by direct or indirect intracranial pressure influence the sense of smell.
2. Inflammatory diseases which involve the olfactory bulbs or the nerve pathways from the bulbs to the brain centers. This group includes malaria, influenza, meningitis, encephalitis, lethargic encephalitis, syphilis, abscess of the brain, extradural abscess and other conditions.
3. Traumas, including basal skull fractures from automobile injuries or industrial injuries, and hemorrhage following an injury or from arteriosclerosis. Skull fracture from an automobile accident has become so common in recent years that it is now a very common cause of some disturbances of the sense of smell.
4. Poisoning, such as that from lead or arsenic.
5. Late manifestations of disease, such as locomotor ataxia and dementia paralytica, in which the disturbance or the loss of the sense of smell is again nothing more than a symptom of the general disease.
6. Edema of the brain following an intracranial surgical operation such as the removal of an orbital tumor by the methods of Dandy, Adson and Benedict.

Chronic Nonspecific Recurring Parotitis. Dr. Carl H. McCaskey, Indianapolis.

Recurring nonspecific parotitis is encountered with sufficient frequency to warrant enumeration of the observations which characterize it, i. e., rapid recurrence, frequent appearance without any apparent cause and association with a discharge of a purulent or milky-colored saliva from Stenson's duct.

Chronic recurrent nonspecific parotitis has a tendency to be painful. The pain may continue from several hours to days or weeks, the gland or glands being moderately enlarged between attacks. At times the enlargement may appear in the morning on arising and disappear after food has been eaten.

Salivary secretion is not usually affected, although there may be an excessive discharge of pus and saliva or more or less constant drooling.

There is, as a rule, little or no febrile reaction, and the patient, whose health is generally unaffected, soon learns to seek relief by light massage over the gland or by removal of fibrin from the duct.

Predisposing causes are not apparent, although surgical operation about the mouth and pharynx has been thought to enter into this condition. Recurring infections of the upper respiratory tract have also been given as an etiologic factor.

On repeated examinations during attacks the gland is usually found to be enlarged or nodular, and on pressure it may be found to be tender. The skin may be red; occasional fluctuations may on incision yield no pus. The duct is not palpable unless it is stenosed, and the mouth of the duct may or may not show inflammatory changes.

The pathologic picture has been described as that of a chronic and subacute inflammation. There is a marked increase of interlobular connective tissue with more or less fibrinous tissue invasion of the lobules; there is marked round cell infiltration with occasional lymphoblasts,

plasma cells, leukocytes and giant cells. In some areas the parenchyma may be replaced by inflammatory tissue.

DISCUSSION

J. WINSTON FOWLKES, New York: It is important to bear in mind the differences between this condition and ordinary infections of the parotid gland. All are familiar with the parotitis which is caused by presence of calculi in Stenson's duct, debilitating diseases, senility or certain forms of psychosis, but the cause of chronic recurring nonspecific parotitis still has a question mark after it.

In conjunction with Dr. McCaskey's comprehensive outline as to the probable causes, I should like to mention again for your consideration two definite findings which seem to be a factor in the majority of cases and deserve more study. There is, first, the striking frequency of instability and mild psychosis in these patients or in close relatives. Second, there is usually some degree of allergy, manifesting itself to a greater or lesser extent as asthma, hay fever, eczema, sympathetic involvement of other glands of the body or achalasia of Stenson's duct.

The treatment during an attack necessarily depends on the findings. If suppuration has taken place, drainage must be established. This can be accomplished by incision or by repeated aspirations with a large needle. Dilation of the duct and frequent massages of the gland are also beneficial.

The patient should be instructed to massage the gland regularly between attacks and to be on the alert for any symptoms which lead to the recurrence of an acute attack, as early establishment of drainage through the duct is highly recommended.

DR. FRENCH K. HANSEL, St. Louis: Dr. McCaskey's patient could have started with an allergy and ended with an infection.

DR. THOMAS E. CARMODY, Denver: In children with this condition my associates and I always expect to find diplococcic infection.

Dilation of the ducts is easy if one is careful and uses probes. We have some probes that Dr. Shea and I have been using for a number of years, with which one can dilate the duct easily and get the secretion.

Of course, in the cases of calculus, as a rule, it is easy to make the diagnosis with a roentgenogram.

As to the treatment in these cases, roentgen radiation relieves the pain and usually the swelling. Most of the lesions do not require opening externally.

We have injected iodized poppyseed oil in a number of cases but always when the parotitis was in a chronic or in a quiescent stage.

DR. ERNEST M. SEYDELL, Wichita, Kan.: Dr. Shea took away a little of my thunder in referring to the method of passing a probe into the duct. I found out a number of years ago that if the mouth was kept open, passing a probe was almost impossible. Before passing the probe I always use a dilator.

I have felt that in these cases the etiologic factor was infection and that the infection then became latent—something similar to a prostatic infection.

Keratosis of the Larynx. DR. H. B. GRAHAM, San Francisco.

This article was published in full in the ARCHIVES OF OTOLARYNGOLOGY (**36**:735 [Nov.] 1942).

DISCUSSION

DR. GEORGE O. CUMMINGS, Portland, Me.: This case report is of importance as it brings vitamin deficiency to attention as a cause for certain laryngeal keratoses. It also suggests that such a deficiency may account for some troublesome cases of sinusitis, tubal congestion, laryngitis, tracheitis and bronchitis in which little in the way of physical findings can be discovered.

DR. CHEVALIER L. JACKSON, Philadelphia: My associates and I have advised limited local removal of areas of keratosis and leukoplakia in the larynx, and in many cases this has been effective. However, I have also seen recurrence with cancerous change, and I believe that dietary correction and vitamin therapy might prevent this.

DR. BURT R. SHURLY, Detroit: This interesting story of what may be accomplished in the human being in cases of vitamin deficiency brings to mind the laboratory work that my associates and I did in the medical school at Detroit with white rats.

One of the first changes in vitamin deficiency is in the vaginal epithelium, which shows keratosis. When this process goes on, a definite series of changes takes place, which are familiar to all as described in the reports of Benay on corneal ulceration, respiratory infec-

tion, ulcers of the tongue, frequent suppuration in the sinuses of the white rat and often suppurative otitis. Those changes, if carried on, terminate in death. We had an interesting monkey in which experimental vitamin A deficiency developed.

We brought out at that time the discovery of halibut liver oil, which is now well known all over the world, but it was produced in our laboratory first and was taken over by various manufacturers of pharmaceutics.

DR. GORDON B. NEW, Rochester, Minn.: I believe that in the treatment of these so-called precancerous lesions the most important thing is a diagnosis based on study of a fresh frozen section at the time of the treatment.

DR. JOHN M. LORE, New York: I am going so far as to state that all keratosis of the larynx should be considered malignant.

As to the question of biopsy technic, when my associates and I are dealing with keratosis of the larynx, we do not have to make multiple attempts at removing an island in the larynx; we have an instrument and a procedure at our disposal which answers the purpose—Kocher's forceps utilized in the stripping operation. There is nothing easier, more efficient and more thorough for removing all that diseased tissue from one cord.

I do not hesitate in any of these cases to do a stripping, either of one or of both sides at a time, or through the anterior commissure instrument.

One of our patients died. It was that patient's case which Dr. Jones presented. The others have been free of symptoms—they go back as far as 1926—with good functional results.

Functional Aphonia. DR. LOUIS H. CLERF and DR. FRANCIS J. BRACELAND, Philadelphia.

Functional aphonia is a hysterical conversion symptom and constitutes an escape mechanism in a personality which is loosely integrated to begin with.

The importance of ruling out organic disease before considering functional disorder is stressed. Not only should there not be evidence of organic findings but there should be positive signs of the hysterical reaction type.

The accompanying voice changes are not due to local laryngeal disease. Mirror laryngoscopy reveals a normal larynx. There is failure to maintain proper adduction of the vocal cords during phonation.

The laryngologist should recognize the essentially psychiatric foundation of this condition and either should equip himself to deal with it or see that the patient is placed under expert psychiatric treatment.

The danger of "fixing the neurosis" by protracted medication and instrumentation is considered, and attention is directed to the fact that mere removal of the superficial symptoms does not mean that the patient is cured. Usually the only result of treatment by electricity and gadgets is a changing of the locus of the complaint.

DISCUSSION

DR. WILLIAM MITHOFFER, Cincinnati: In examining the larynxes of patients afflicted with functional aphonia, one sees that an attempt is made to adduct the cords but, because of lack of will power, the cords fail to meet in the midline. When abduction is attempted, a quivering motion is often detected. Occasionally, in long-standing cases, the patient presses sufficiently hard and brings into play the false vocal cords. This phonatory use of the false cords may overshadow the functional aphonia and make the treatment more difficult and prolonged.

My method of treating these patients is as follows: They are first taught diaphragmatic breathing, and this is readily done by placing a band across the lower part of the thorax, in the region of the diaphragm, and then telling the patient to take a deep breath and attempt to stretch the band. After the patient has done this several times, he is told to hum the consonant "m," and is taught to use prolonged expiration with retraction of the abdominal muscles during expiration. When proper chest tones are emitted by humming the consonant "m," vowels may be added. Pressure with the fingers on the thyroid cartilage at this stage of the treatment is of assistance in restoring the voice. I am thoroughly convinced that the breathing exercises outlined play a most important role in the treatment, and in mild cases this form of treatment is all that I have found necessary. The patient is instructed to use the humming exercises fifteen to twenty times three times a day and after each exercise period to read aloud with proper tone placement ten minutes at a time, for at least a month following restoration of the voice. It is not too much to say that most of the patients with functional aphonia respond to this form of simple treatment. In long-standing cases faradic stimulation may be found necessary. When faradic current is used, the patient is told that the use of the current will restore the voice. I do not agree with the essayist that the patient

should be told that "the use of elecrotherapeutic agents is only an adjunct to the main therapy." The electrodes are placed in the region of the thyroid cartilage; the patient is asked to use humming exercises, and the current is applied, beginning with a weak stimulation which is slowly increased, if necessary, until it becomes painful.

The various paresthesias of the throat are the earliest manifestations of phonasthenia, and they disappear when the patient is taught proper tone placement. I am not convinced that a diagnosis of psychophonasthenia can be made because of the presence of paresthesia without testing the tone placement of the voice. If the patient is not using the voice correctly, phonasthenia is, in my opinion, without doubt the cause of the symptoms. If the voice is correctly used in the presence of paresthesia, other causes, such as lateral pharyngitis, must be eliminated from the picture before a diagnosis of psychophonasthenia is made. It is true, paresthesia of the throat may occur in hysterical patients without the presence of phonasthenia. In these patients hysteria, with or without aphonia, and not psychophonasthenia, would be the correct diagnosis.

Dr. James S. Greene, New York: Dr. Clerf calls attention to a type of disorder which should be of special interest to physicians in a world at war, since hysterial manifestations of one type or another will undoubtedly be on the increase during the next few years, particularly among men in the front line trenches. An incapacitating hysterical symptom enables the subject to avoid danger without risking the stigma of cowardice. He is safe—without dishonor.

Psychophonasthenia seems to represent a breakdown of the personality under cumulative stress, whereas aphonia is an escape from some specific threat. Psychophonasthenia is never a simple escape; in fact, it is a complex disorder, evidenced by the fact that one never sees —at least not to my knowledge—a spontaneous resolution of the condition. On the other hand, in hysterical aphonia spontaneous resolution is not at all uncommon.

It is also interesting to note that the psychophonasthenic person is usually of superior intelligence, while the hysterically aphonic patient, as one might suspect from the very simplicity of the mechanism he uses to escape, is more frequently of average or even below average mentality. The personality of the aphonic patient as a whole is much less mature and less efficient than that of the patient suffering from psychophonasthenia. Although both disorders represent a conversion of psychic conflicts into somatic symptoms, the essential mechanism in psychophonasthenia is one of trying to adjust to the demands of reality, while in the case of the hysterically aphonic patient the essential mechanism is one of flight from making even an attempt at adjustment.

Dr. Frank M. Sulzman, Troy, N. Y.: In 1935 a young married woman was riding in a private vehicle which collided with one of the public buses in Troy. As a result of that accident, her head struck the roof of the car. There were various injuries, and there was a loss of voice.

Her family doctor had me see her several times after a few weeks. Examination showed no lesion, but the best voice I could get was a very modified whisper. I so reported to this doctor. I saw her at different intervals for a period of two years.

Six years after that a general surgeon had to operate on her for a pelvic condition. I happened to be in the hospital a few days after her operation and a nurse who knew I had taken care of this patient said, "Doctor, your patient has regained her voice. Coming out of the anesthesia, her voice became perfectly normal and she made the usual sounds of a patient after having anesthesia."

Dr. Gabriel Tucker, Philadelphia: It is sometimes difficult to get a person who has been aphonic for a period of five years to produce a tone. Such patients have convinced themselves that they cannot produce a tone. It is a matter of suggestion, and I think it is important to get them to produce a clear tone.

My associates and I have used the method—I am sure you have used the same method—of introducing the direct laryngoscope into the larynx without cocainization. It will prevent the aproximation of the ventricular bands, and the patient can be got to phonate with the laryngoscope in position.

If one has a patient in whom the larynx goes into spasm and who cannot prevent the two cords from coming together, he will produce a tone, after which one can carry out the suggestion with the aid of the psychiatrist.

Cysts of the Floor of the Mouth. Dr. W. H. Johnston, Santa Barbara, Calif.

The cysts usually found in the floor of the mouth are: dermoid, epidermoid, ranular, thyroglossal, mucous and branchial.

The true nature of ranula is still disputed, and more investigation is necessary to explain its genesis satisfactorily.

Laryngologists are often the first to discover or to be consulted regarding the diagnosis and treatment of these lesions.

Surgical removal is accepted as the most successful method of treatment.

DISCUSSION

DR. THOMAS E. CARMODY, Denver: The dermoid is usually found in the anterior part of the tongue or just above the mylohyoid muscle and sometimes progresses backward if it is allowed to grow to any great extent.

The mucous cyst is frequently diagnosed as ranula. As Dr. Johnston has said, "ranula" is a term that is applied frequently to any swelling in the floor of the mouth, but with true ranula one usually sees a gelatinous substance in the sac.

The sublingual cyst is also easily diagnosed. It is found usually as a true cyst; it may be, however, simply a damming back by a stricture or a foreign body in Wharton's duct.

DR. CHARLES T. PORTER, Boston: My associates and I do not see as many branchial or retention cysts as we used to see ten years ago. One might conclude that oral hygiene and increasing education along that line have something to do with this disease disappearing, at least in our locality, and that the cysts are—certainly many of them—dependent somewhat in their origin on local infection and local caries.

The treatment is primarily excision, and according to some authors one would imagine this is a perfectly easy thing to do. It is comparatively easy with the thick-walled dermoid and epidermoid cysts. The branchial cyst is not so easy. With the thyroglossal cyst many have had failures and finally have had to take out part of the hyoid bone and resect the cecum of the base of the tongue. I have not found it easy to dissect the thin-walled cyst, particularly if it has lasted for any length of time, is of any size or has a thin wall. I have resorted to the multilocular or the inversion fistula operation, which I think is almost as good as excision.

I do not quite agree with the author that it is dangerous to aspirate these cysts. I think that frequently one can learn a great deal from careful aspiration and perhaps introduction of lipoiodine for a roentgen examination, thereby finding out the extent of the cyst, whereas otherwise one would be getting at some of its ramifications.

Acute Mastoiditis: 1931-1941. DR. LAWRENCE R. BOIES, Minneapolis.

The decade just past has witnessed two events which must rank in importance with any previous milestones in the progress of otology. One was the development of knowledge regarding suppuration within the petrous portion of the temporal bone. The other arrived with the application of sulfonamide compounds to the middle ear and mastoid presenting acute suppurative disease and its complications.

Approximately eleven years have passed since American otologists first became interested in petrositis. About five years ago chemotherapy entered the otologic picture.

This article is essentially an effort at taking inventory at this time on these two occurrences and has as a background a series of 650 cases of acute mastoiditis requiring surgical intervention during the years from 1931 to 1941.

The quality of mastoid surgery has been improved as a result of the development of knowledge regarding suppuration in the temporal bone. In consequence the incidence of postoperative complications will decrease.

The use of the sulfonamide compounds has decreased the incidence of acute otitis media and acute mastoiditis, particularly in patients who have had early and skilled medical care. It is reasonable to believe, however, that a share of the decrease in the past two or three years is due to other causes, principally to the lack of, or the better control of, the epidemic infections which cause these diseases.

The use of the sulfonamide compounds has markedly lowered the mortality from otitic sepsis and otitic meningitis. Experience is reported with 23 cases of otitic sepsis with a mortality rate of 4.4 per cent. This represents a 75 per cent improvement in the rate from this cause. A mortality rate of 20 to 30 per cent in 15 cases of otitic meningitis is reported. This disease was formerly practically always fatal.

DISCUSSION

DR. ROBERT L. MOORHEAD, Brooklyn: In the Brooklyn Eye and Ear Hospital in the years from 1931 to 1941 3,384 patients were operated on for acute mastoiditis, and during that time there were 37 in whom the petrous pyramid was operated on and pus excavated.

Thus in Dr. Boies's series the proportion of the incidence of infection of the pyramid to that of acute mastoiditis was about 1 to 65 whereas in Brooklyn the proportion was about 1 to 91, and even this is misleading for many of the patients with petrositis were referred to me from other localities or other hospitals where the diagnosis had been made; they were sent in simply for operation. Thus the proportion was actually much lower than 1 to 100. Of these 37 patients, only 8 were found to have a perilabyrinthine tract of cells leading into the pyramid. This was in spite of a thorough search—for in the operation which we use all of the bone of the mastoid is removed down to the level of the labyrinth and then the semicircular canals are skeletonized, so that the possibility of a tract being overlooked is rather remote. In the last few years (1939-1942) we have had comparatively few cases of petrous infection, but I believe that this is due to the much diminished incidence of mastoiditis rather than to the more thorough operation as suggested by Dr. Boies. For years we have been rather particular in cleaning out the petrosal or so-called solid angle down to the labyrinth, and I have failed to notice any particular change in this since the recognition of petrositis as a clinical entity. Be that as it may, the important point stressed by the paper is the necessity of thorough exenteration of all cells down to the labyrinth and the drainage of any areas that are found extending around or beyond the semicircular canals. As I described in my reports to this society in 1939 and 1941, I do not believe it necessary to do radical mastoidectomy in order to drain the pyramid. After recovery the patient is left in most cases with a normal middle ear and good hearing.

Chemotherapy. Dr. Lawrence R. Boies, Minneapolis.

The exact status of chemotherapy is still undetermined, although the prevailing opinion in my section of the country is that it has reduced both the incidence and the complications of acute mastoiditis.

DISCUSSION

Dr. Theodore E. Walsh, St. Louis: Dr. Boies believes in the early administration of the sulfonamide compounds in otitis media. His most interesting chart shows the reason for this belief and is worthy of study. The remarkable drop in the incidence of mastoiditis since 1937 in those cases in which the sulfonamide compounds were given early as compared with those in which the drug was withheld until late is proof of their value. I think our experience in this regard bears out Dr. Boies.

My associates and I have observed as many cases of mastoiditis this year as in previous years, but our patients, like those in the Boston Children's Hospital, have not had early chemotherapy.

I have heard men argue that they do not like to give the sulfonamide compounds in every case of otitis media because in the past many patients got well without these drugs. However, I doubt the ability of even the keenest clinician always to tell in which case of otitis media mastoiditis will follow and in which it will not.

I should like to enter a plea against the use of the term "masking" in relation to the sulfonamide compounds. The whole picture of otitis media and mastoiditis has changed in these last years. One can no longer rely on the old textbook signs of mastoiditis as they were before the advent of chemotherapy. One must now learn the signs and symptoms of the otitis media–sulfonamide compound syndrome. The indications for surgical intervention are still present but are not as clearcut as they were in the past. Consideration of the possibility of signs being masked by sulfonamide compounds might lead to these agents being withheld, which I think would be a great pity. It is probable that in their effects on the complications of mastoiditis the sulfonamide compounds have demonstrated their greatest value.

Hartmann has shown in a publication now in press that with the advent of these drugs the mortality rate for thrombosis of the lateral sinus has dropped from 35 to 10 per cent, and that the mortality rate for meningitis due to the hemolytic streptococcus, previously invariably fatal, is now but 11 per cent and that for meningitis due to the pneumococcus 63.7 per cent.

In cases of meningitis physicians are no longer anxious to resort immediately to surgery but prefer to give twenty-four to thirty-six hours of preoperative medication with the appropriate sulfonamide compounds. The blood level is raised as fast as possible by administering the drug intravenously as well as by mouth, and this is perfectly safe provided there is adequate administration of fluid at the same time.

Dr. Burt R. Shurley, Detroit: In the last series of 10 cases of acute mastoiditis with all the indications for operation present, the usual blood count and roentgen examination having been made, we have been able to avoid operation by the use of chemotherapy with the proper choice of sulfonamide compounds and by their proper and continued dosage.

These patients must be in the hospital, however, just as if they were operative patients, because the science and art of this medication and the choice of the variety of the seventeen different preparations that are offered to one are of the greatest importance.

Sulfadiazine has been by far the most satisfactory in my hands.

Local Use of Sulfonamide Compounds, Gramicidin (Tyrothricin) and Penicillin in Otolaryngology. Drs. John E. Bordley, S. J. Crowe, David A. Dolowitz and Kenneth L. Pickrell, Baltimore.

We find that in most cases infection of the ears or the sinuses is secondary to acute coryza or nasopharyngitis. Controlled study of such cases indicates that many of them may be prevented by spraying the nasal passages and the pharynx with a 2.5 per cent sulfadiazine-triethanolamine solution. The formula is as follows:

June 1 to 3, 1942

Sulfadiazine powder	2.5 Gm.
Triethanolamine	7.5 cc.
Butyl parahydroxybenzoate (butoben, Merck)	0.05 Gm.
Boiled distilled water to make	100.00 cc.

The distilled water is boiled and cooled to 90 C. To a portion of this the triethanolamine and butyl parahydroxybenzoate is added. The sulfadiazine is then dissolved in this solution by agitation. The solution is cooled and filtered, and sufficient boiled distilled water is added to make up the desired amount.

This mixture is readily absorbed and appears in the blood stream, but since not more than 20 cc. (0.5 Gm. of sulfadiazine) is used in any one day, the concentration in the blood is not high. The concentration of sulfadiazine in the respiratory mucosa has not been determined, but other studies indicate that it may be six times greater than that in the blood.

Patients with coryza or nasopharyngitis were alternately placed in a "treated" and a "control" group. All were treated alike except that the controls were sprayed with the solvent (triethanolamine) and the treated ones with the solvent plus 2.5 per cent sulfadiazine. From 1.5 to 2 cc. of sulfadiazine solution was used at each treatment. Usually eight such treatments were given daily for the first two days and thereafter five or six treatments a day for three days. Patients in both groups were given acetylsalicylic acid and codeine when necessary for pain and elixir of terpin hydrate for cough. In addition to the spray, a 5 per cent sulfadiazine ointment was applied to the nasal vestibule twice daily; in the controls the base only was used. All were given neo-synephrin hydrochloride solution for nasal obstruction.

The control group usually experienced subjective improvement on the second day. Bacteriologic studies showed a decrease in the number of colonies of the beta hemolytic streptococcus in cultures of material from the pharynx for patients in the treated group as compared with the control group, but alpha and gamma strains were unaffected. The most constant and immediate benefit of the sulfadiazine spray was the prevention and control of the irritating cough so often accompanying nasopharyngitis. None of the treated patients required terpin hydrate, while spasmodic coughing attacks lasting five to fifteen days occurred in 9 of the 14 controls. One control patient with red, edematous pharynx, postnasal discharge and persistent cough for eight days was transferred to the treated group; all symptoms disappeared in forty-eight hours.

Two of the 16 treated patients had slight clouding of one antrum on transillumination, which cleared without local treatment; 7 of the 14 controls had complicating infection of one or more accessory sinuses. One treated patient had had acute sinusitis with each cold for the preceding four years, six such attacks requiring irrigation of an antrum during the previous winter. The current absence of sinus infection was attributed to the use of the sulfadiazine spray.

Use of the spray is contraindicated in those persons (about 3 per cent) in whom vasomotor rhinitis arises because of sensitivity to either the drug or the solvent.

Tyrothricin (a crystalline substance isolated from soil bacilli by Dubos in 1939) and penicillin are powerful bactericidal agents for most but not for all organisms. They usually destroy anaerobic and the Lancefield beta streptococci, a group of organisms resistant to sulfonamide compounds. To be effective tyrothricin must come in contact with the infecting bacteria; it cannot be used intravenously or subcutaneously. Its chief value in otolaryngologic practice is in the prevention of postoperative wound infection. It is almost totally insoluble in water and must be used as a suspension. Applying it freely to exposed bone and surrounding soft parts after an operation on infected nasal sinuses or mastoid and saturating the packs with a 1 per cent suspension of tyrothricin in poppyseed oil add greatly to postoperative comfort and shorten hospitalization.

Penicillin is more valuable than tyrothricin. It is soluble in water, nontoxic and is absorbed by the tissues. It is often more effective than sulfonamide compounds or tyrothricin against staphylococci and is equally effective against aerobic and anaerobic streptococci and pneumococci. Penicillin is not available at present in the refined form suitable for intravenous administration, but it may be made by growing the mold Penicillium on the surface of suitable fluid mediums. When the end point is reached, as determined by biologic tests, the p_H and the color, the fluid on which the fungus was grown is filtered through porcelain to remove all mold and bacteria. The filtrate is suitable for local application in infected wounds and for irrigation of middle ear or accessory nasal sinuses. It should be kept sterile and in a refrigerator, preferably frozen. Proteus and some other gram-negative organisms grow freely in a penicillin solution.

Since most chronic infections of the upper respirtory tract are due to a variety of organisms, intelligent use of these bactericidal agents demands accurate identification of infecting bacteria and biologic tests to determine their sensitivity.

Changing Conception of the Management of Chronic Progressive Deafness. DR. FREDERICK T. HILL, Waterville, Me.

Otologists realize the impossibility of curing deafness. All of their therapeutic efforts finally come down to prevention, correction by circumvention or amplification, and rehabilitation. The changing of the conception of the management of chronic progressive deafness through these years is a result of better understanding of the physiology of hearing and of the pathology of deafness and the development of precision methods of testing. Many of the older procedures have been retained; indeed some of those discarded have been revived as their once theoretic premises have been substantiated by modern research. Otologists recognize the effect of tubal obstruction and of negative pressure and try to remedy the cause by local treatment, by surgical operation on the nose or throat or by irradiation of the involved tissues. They keep in mind the possibility of the absorption of toxins and strive to eradicate foci of infection when indicated. They consider the effect of occupation or environment. They realize the importance of good general health and advise the correction of any nutritional or endocrine deficiency. And, above all, they urge the need for early recognition and correct diagnosis if prevention is to be effective. For the majority of patients with established deafness sufficient to be handicapping, correction by amplification will be preferable. Operative treatment may be suggested to those who, after critical study, meet all the requirements laid down as essential to successful fenestration, provided they fully understand its present status. And finally otologists recognize the importance of cultivating a personal interest in each patient, adapting treatment to individual circumstances and conditions and endeavoring in all possible ways to assist the patient to achieve an improved social and economic life.

DISCUSSION

DR. WALTER HUGHSON, Abington, Pa.: Peculiarly enough, the most dramatic form of therapy now available—fenestration of the labyrinth—has never been satisfactorily explained on any physiologic basis.

There is no question that precise and thorough audiometric studies require time and patience, but the results are rewarding both to the otologist and to the patient. Air conduction audiograms must be made at frequent intervals. My associates and I have shown experimentally that bone conduction can be modified and changed by numerous factors and that bone conduction itself is not the final diagnostic factor in nerve deafness. Fortunately we now have available two diagnostic methods which are absolutely differential, namely, the loudness balance and the fatigue test. Further, as a preliminary to the total therapeutic effort, the patient's actual measurable disability must be determined by controlled speech reception studies. When all of this has been done and is a matter of record, we can approach the problem of a comprehensive survey of the total constitutional situation. Here again time and care are essential if the effort is to be of any possible consequence. Thorough local examination, general physical examination and sympathetic psychologic study undoubtedly all take time but are an essential prelude to any therapy.

The surgery of deafness is an alluring subject. In it undoubtedly lies much of the future of otologic therapy. I mention the round window graft operation only to correct a false impression which Dr. Hill's remarks may have made. Before operation each patient in our small series had been given elsewhere the diagnosis of otosclerosis. We have previously stated that when this is the case the operation should further depress the hearing. This has not occurred in any of these cases. It can be assumed, therefore, that either the original diagnosis was incorrect in every instance or that fixation of both windows in the human being does not eliminate hearing by air conduction.

Finally the problem of the fitting of a hearing aid must be considered. It is the only treatment with which we can definitely promise help. The results are immediate and permanent. Hearing aids are fitted, not prescribed. Regardless of therapy, every patient with a hearing impairment of sufficient extent should be in a position to resort to a hearing aid in case of necessity. We should all be familiar with the technical problems involved.

Control of Head Noises: Their Illusions of Loudness and Timbre. Dr. Edmund P. Fowler, New York.

This article was published in full in the Archives of Otolaryngology (**37**:391 [March] 1943).

Unilateral Vasomotor Rhinitis Due to Interference with the Cervical Sympathetic System. Edmund Prince Fowler Jr., New York.

This article was published in full in the Archives of Otolaryngology (**37**:710 [May] 1943).

Mixed Tumor of the Parotid Gland in the Tonsillar Area. Dr. Carl W. Lupo, Brooklyn.

H. E. McC., 38 years old, was admitted to the Brooklyn Eye and Ear Hospital Dec. 3, 1935. A few weeks before admission a medical examiner for an insurance company reported that the patient had an abscess, ready for incision, in his right tonsil. When the examinee remarked that he had no sore throat, further examination was suspended pending treatment and report by his family physician. Retrospectively the family had noticed that his voice had had a nasal tone for a number of years.

Physical examination revealed a hard cystlike growth occupying the entire right tonsillar area. It extended upward into the pharyngeal wall to about the level of the soft palate, which was freely movable from the tumor; the adjacent tissues were reddened, which probably was the reason for the earlier assumption of tonsillar abscess.

With the patient under general anesthesia, a hard, dense, cartilage-like ovoid tumor about 5 cm. in diameter was excised from the right tonsillar area, devoid of capsule and more or less held in its bed by a mass of fibrous bands, some of which were stringy and extended into the deeper layers of the lateral pharyngeal wall.

When the tumor was extracted, a hypertrophied and infected tonsil was found to have been pushed downward to about the level of the epiglottis. The tonsil was encapsulated and entirely distinct from the tumor. The bed of the tumor was closed with black fiber sutures. Tonsillectomy was performed also on the left. No other signs of tumor were found.

The pathologic diagnosis was as follows: (1) mixed tumor of the parotid gland with epithelial structures set in a matrix varying from myxomatous tissue to cartilage and bone; (2) chronic infection and lymphoid hyperplasia of the tonsil. No tumor tissue was found in the tonsil.

Recent examination shows no recurrence.

News and Comment

AMERICAN OTORHINOLOGIC SOCIETY FOR ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY

The first annual meeting of the American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc., will be held Friday, November 12, in New York. The clinic and business meeting will be in the morning and afternoon at the Manhattan General Hospital, and the scientific meeting, at 8 p. m. at the Academy of Medicine. The speakers at the scientific session will be Drs. Romeo Luongo (president), Samuel Fomon, Samuel J. Kopetzky, Alfred Schattner and Arthur Proetz. All communications should be addressed to the secretary, Dr. Jacob Daley, 104 East Fortieth Street, New York.

CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION

A sum of money having accrued from the Casselberry Fund of the American Laryngological Association, a prize will be offered in 1944 for original investigation in the art and science of laryngology or rhinology. Theses must be in the hands of the secretary, Dr. Arthur W. Proetz, 1010 Beaumont Building, St. Louis 8, Mo., before March 1, 1944.

CORRECTION

In the article by Dr. Samuel Salinger entitled "The Paranasal Sinuses," in the October issue (ARCH. OTOLARYNG. **38**:373, 1943), the dosage in the last line on page 376 should read "1 Gm. daily for four days" instead of "daily doses of 1 grain (0.065 Gm.)," and the second sentence at the top of page 377 should read "He comments favorably on the use of a pack saturated with 1 per cent solution of cocaine hydrochloride in connection with the infra-red lamp."

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Claude C. Cody, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.

American Academy of Ophthalmology and Otolaryngology

President: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
President-Elect: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis, Mo.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Place: Chicago. Time: Oct. 10-13, 1943.

American Broncho-Esophagological Association

President: Dr. Robert L. Moorhead, 125 Ramsen St., Brooklyn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

SECTIONS:

Eastern.—Vice President: Dr. Westley M. Hunt, 907-5th Ave., New York 21.
Southern.—Vice President: Dr. William C. Warren Jr., 478 Peachtree St. N. E., Atlanta, Ga.
Middle.—Vice President: Dr. Fred W. Dixon, 2060 E. 9th St., Cleveland.
Western.—Vice President: Dr. Simon Jesberg, 500 S. Lucas Ave., Los Angeles.

American Otological Society

President: Dr. Wesley C. Bowers, 17 E. 61st St., New York.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.